Author:
Eastwood, Andy

Title:
Alcohol related aggression

exploring the acute and chronic effects of alcohol consumption on key aspects of social cognition

Alcohol related aggression: exploring the acute and chronic effects of alcohol consumption on key aspects of social cognition

Andrew P.R. Eastwood

University of Bristol
School of Psychological Science
December 2020

A dissertation submitted to the University of Bristol in accordance with the requirements for award of the degree of Doctor of Philosophy (PhD) in the Faculty of Life Sciences

Word count: 38,586

ABSTRACT

Research indicates that alcohol consumption causes aggressive behaviour, but it is not an inevitable consequence. It is likely that alcohol increases aggression by impairing cognitive and perceptual mechanisms associated with the behaviour. The purpose of this thesis was to extend the current understanding of alcohol-related aggression, by investigating the acute and chronic effects on emotional face perception and social interaction interpretation. Both experimental and cross-sectional approaches were used. As emotional expressions are a form of non-verbal communication likely to influence behaviour, the effects of acute and chronic alcohol consumption on recognition were explored. It was anticipated that acute consumption would impair emotion recognition. Alcohol dependence is linked to emotion processing deficits and it was anticipated that chronic consumption would similarly predict poorer recognition. Next, the influence acute alcohol consumption has on hostile interpretations of emotional expressions was explored. Hostile attribution bias is linked to increased aggression and evidence from forensic samples highlights that aggressive individuals display emotion processing deficits. It was anticipated that alcohol would cause a similar profile of impairment. This thesis then explored whether acute alcohol influenced impressions formed when viewing dyadic social interactions. These involve two people interacting and are likely to influence the perceiver's behaviour. It was anticipated that alcohol would influence the impressions formed when viewing dyadic social interactions. Results showed that acute alcohol consumption reduced global emotion recognition and impaired sad and fearful expressions recognition. Chronic consumption also impaired the ability to recognise sadness. Acute consumption also resulted in happy faces being interpreted as more hostile. The impressions formed when viewing dyadic interactions were not influenced by acute alcohol consumption. As cues of submission (i.e., sadness and fear) are influenced by acute and chronic consumption, and happiness is interpreted as more hostile when intoxicated, this may lead to increased aggression.

ACKNOWLEDGEMENTS

This has been by far the easiest section of my thesis to write! I would like to thank my incredible supervisory team at the Tobacco and Alcohol Research Group (TARG), University of Bristol. Angela Attwood, Ian Penton-Voak and Marcus Munafò have been exceptionally supportive and suitably critical throughout my PhD journey. I'd also like to say an extra thank you to my primary supervisor Angela. I have learnt a great deal under her careful guidance, and I owe a lot of my successes so far in my career to her. I would also like to thank the 'Upstairs Bears' (formally the 'G2 Crew') for being an incredibly supportive research network and a great bunch of friends. The office discussion over coffee, as well as the endless amounts of sweets and treats have made these last four years a breeze.

It always pays to have a supportive family around you and mine have definitely been there when I've needed them. I'd like to dedicate this work to my parents Karen and Tracy. Their emotional and financial support have given me a platform to build upon and without them I would not have reached this stage in my career. To my brother Sam and sister Louise, I am grateful for the laughs and jokes when I needed distracting. To succeed in life, everyone needs a friend like Craig. Thank you for always being on the end of the phone, and for always reassuring me when I start doubting my career decisions! Your nuggets of wisdom have truly helped shape this work. I would also like to thank my wonderful dogs Frank and Poppy for keeping me fit and keeping me popular during my studies. Dogs really do give you that boost when you need it most. Finally, I would like to thank my fantastic fiancée Ellis who honestly deserves a medal for putting up with me throughout this journey. Your kindness, love and support has helped keep me on track. This most certainly would not have been possible without your encouragement. We make a pretty good team!

AUTHOR’S DECLARATION

I declare that the work in this dissertation was carried out in accordance with the requirements of the University's *Regulations and Code of Practice for Research Degree Programmes* and that it has not been submitted for any other academic award. Except where indicated by specific reference in the text, the work is the candidate's own work. Work done in collaboration with, or with the assistance of, others, is indicated as such. Any views expressed in the dissertation are those of the author.

SIGNED: DATE:

TABLE OF CONTENTS

LIST OF FIGURES

LIST OF TABLES

CHAPTER 1: INTRODUCTION

1.1. Chapter Overview

This thesis of work focuses on the relationship between alcohol consumption and aggression. Aggression is defined as “any form of behavior directed toward the goal of harming or injuring another living being who is motivated to avoid such treatment” (Baron & Richardson, 1994; page 7). Important components of aggressive behaviour are the immediate intention to cause harm, the perpetrator’s awareness that the action will cause harm, and the belief that the victim is motivated to avoid the behaviour (Anderson & Bushman, 2002; Baron & Richardson, 1994; Geen, 2001). Prior research has indicated that this relationship is causal (Beck & Heinz, 2013; Chermack & Giancola, 1997; Hoaken & Stewart, 2003) and the exact mechanisms by which consumption causes aggressive behaviour are complex. One mechanism that is relatively overlooked in the literature is the role of emotional face expression processing (Attwood & Munafo, 2014).

Facial expressions are a key form of non-verbal communication (Moriya et al., 2013) and alcohol induced deficits may contribute to explaining why certain individuals become more aggressive when intoxicated (given that aggression is not an inevitable consequence of consumption). There is some evidence that suggests that both acute and chronic alcohol consumption influences emotion recognition. Chronic consumption specifically in alcohol dependent individuals is linked to emotion processing deficits (Kornreich et al., 2001; Maurage, Campanella, Philippot, Martin, et al., 2008). It is however unclear whether non-dependent drinkers (i.e., individuals that consume alcohol socially) also demonstrate impaired emotion processing ability. Acute alcohol consumption has been found to reduce overall emotion recognition (Tucker & Vuchinich, 1983), as well as the processing of specific emotions (Attwood, Ataya, et al., 2009; Attwood, Ohlson, et al., 2009; Dolder et al., 2017; Khouja et al., 2019). However, there is no clear

consensus on how acute consumption impacts the processing of individual emotional expressions. Some evidence suggests that alcohol enhances the processing of positive emotions such as happiness (Dolder et al., 2017), whereas other work suggests that alcohol disrupts the processing of sadness (Craig et al., 2009) and increases anger biases (Khouja et al., 2019). Another important line of inquiry surrounds the interpretation of emotional facial expression (i.e., how they are evaluated). Hostile attribution bias is considered to be a key contributing factor in aggressive behaviour (Crick & Dodge, 1996). Evidence from forensic and clinical samples suggests that individuals prone to aggression demonstrate hostile biases when perceiving emotional expressions (Smeijers et al., 2017). It is unclear whether alcohol consumption results in a similar profile of interpretation bias. Social interactions are complex and often involve forming impressions of more than one individual. This perceptual information taken from these third party encounters can impact the success of a social interaction (Quadflieg & Penton-Voak, 2017). Evidence suggests that these impressions can directly influence the perceivers own behaviour (Quadflieg & Westmoreland, 2019). It is currently unclear whether alcohol influences dyadic social interaction (i.e. interaction between two people) impression formation.

This thesis aims to address the following: Chapter 2 focused on exploring the effects of alcohol consumption on emotional face processing in social alcohol drinkers who were either high or low in trait aggression. It was hypothesised that there will be a global deficit in emotion processing, an increased sensitivity towards perceiving anger, and a decreased sensitivity towards perceiving sadness following alcohol compared to placebo. These effects were anticipated to be more pronounced in high compared to low trait aggressive drinkers. Chapter 3 aimed to test whether chronic alcohol consumption is associated with poorer emotional face recognition in a non-dependent sample. It was also anticipated that greater weekly alcohol consumption and

frequent binge drinking over a minimum period of 5 years would disrupt the processing of specific emotions. Chapter 4 focused on exploring the effect of acute alcohol consumption on hostile attribution bias of emotional facial expressions. It was hypothesised that there will be greater hostile attribution bias towards emotional facial expression following acute alcohol consumption compared to placebo. Chapter 5 aimed to test whether acute alcohol consumption affects hostile evaluations of dyadic social interactions (i.e., two people interacting). It was hypothesised that dyadic social interactions will be seen as more hostile following acute alcohol consumption compared to placebo.

1.2. Alcohol Consumption and Aggressive Behaviour

A large body of literature has investigated the influence of acute and chronic alcohol consumption on various types of aggressive behaviour (Pihl & Sutton, 2009). These include intimate partner violence (Foran & O'Leary, 2008), interpersonal violence towards strangers (Cogan & Ballinger, 2006), sexual aggression in males (Testa, 2002), and the perpetration of violent crime (Darke, 2010; McClelland & Teplin, 2001; Murdoch et al., 1990). General alcohol consumption has been shown to be a risk factor in interpersonal violent offences in forensic samples (Lundholm et al., 2013). These authors highlight that 49% of remand prisoners reported consuming alcohol within the 24 hours prior to their violent crime. The associated risk of violence was found to increase when adjusting for amount of alcohol consumed. These conclusions are well supported and consistent with similar forensic sample research that highlights the role of alcohol consumption in the perpetration of interpersonal violence (Haggard-Grann et al., 2006; Hoaken & Stewart, 2003). In general population samples, prolonged habitual drinking is positively linked to the perpetration of aggressive acts (Scott et al., 1999; Wells et al., 2000). It is clear that excessive alcohol consumption is associated with a wide range of aggressive behaviours and violent acts.

This highlights the importance of considering how prevalent these occurrences are in both national and global populations.

1.2.1. Alcohol Related Aggression Prevalence

In the United Kingdom, the Office for National Statistics (2019) report that 39% of violent incidences were committed by adults under the influence of alcohol (most recent data recorded between April 2017– March 2018). Of these, 44% of cases were deemed to be violence that led to injury and 36% were considered to be violence that did not lead to injury. This report also shows that 49% of the victims were not known to the perpetrator, 35% were known, and 31% were cases of domestic violence. National data also show that 57.1% of alcohol related violent incidents were committed in a pub, club or on the street (Office for National Statistics, 2018). A series of population level studies similarly highlight the frequency of aggressive offences that involve alcohol intoxication at the time of the event. A study using a Canadian sample report that alcohol was involved in 38.1% of serious arguments, 56.5% of threatening behaviour, and 67.9% of incidents of physical aggression (Wells et al., 2000). These authors conclude alcohol intoxication at an event level (i.e., consumed at the time of the incident) plays an important role in aggressive behaviour. They also note that intoxication was more associated with physical than verbal aggression. Bye (2007) conducted a time-series analysis of population level data in Australia. They reported that an increase in alcohol consumption of one litre per person per year predicted an increase in violence of around 8% demonstrating a strong link between increased consumption and aggressive behaviour at a population level. A study of United States college students reported that alcohol consumption co-occurred with general aggressive behaviour in 9% of cases, and in 28% of sexual aggression cases (Stappenbeck & Fromme, 2010). Global estimates suggest that alcohol consumption is involved in approximately half of violent crimes (Darke, 2010) and sexual assaults

(Testa, 2002) worldwide. These studies and population level data are compelling, but only offer insight into the frequency of aggression under the influence of alcohol. These studies also rely heavily on memory recall of the event and are subject to error and bias which impact the credibility of the self-reported accounts. These studies also lack the ability to address causal inferences such as how acute consumption of alcohol increases the propensity to act aggressively. It is therefore important to consider laboratory studies that are able to experimentally manipulate alcohol consumption and directly measure aggressive responding (Buss, 1961; Taylor, 1967). This allows causality to be addressed and other factors such as individual differences in personality, typical drinking behaviour and other demographic information to be controlled for.

1.2.2. Alcohol Consumption Causes Aggressive Behaviour

Evidence suggests a link between both acute and chronic alcohol consumption and aggressive behaviour (Beck & Heinz, 2013; Chermack & Giancola, 1997; Hoaken & Stewart, 2003). There is a wealth of support for this conclusion from several quantitative and qualitative literature reviews (Bushman & Cooper, 1990; Ito et al., 1996; Lipsey et al., 1997). One of which, a meta-analysis of over 30 experimental studies, concluded that this association was causal (Bushman & Cooper, 1990). These authors report an effect of alcohol on aggressive behaviour when an alcohol vs. placebo (i.e., non-alcoholic drink administered as alcoholic) comparison was made. This comparison allows the influence of expectation to be controlled. However, they also report no effect of alcohol on aggression when an anti-placebo (i.e. alcoholic drink administered as non-alcoholic) vs. control (i.e., a non-alcoholic drink administered as non-alcoholic) comparison was made; which would best model a pure pharmacological effect. They therefore concluded that the effect of alcohol consumption on aggressive behaviour was not solely pharmacological but likely to be influenced by psychological factors. More recently, Duke et al.

(2018) investigated 32 meta analytic studies that reviewed experimental, longitudinal and cross-sectional studies exploring the link between alcohol and aggression in a large 'meta-meta-analysis'. These authors concluded that the effect of alcohol on aggressive behaviour was medium (d=0.39). There is overwhelming support for a causal relation.

While the general consensus in the literature is for a positive causal relationship (i.e., alcohol is a contributing cause of aggression), aggressive behaviour is by no means an inevitable consequence of alcohol consumption as not everybody that consumes alcohol becomes violent or experiences heightened levels of aggression. This suggests that the influence consumption has on aggressive behaviour is not purely pharmacological. Instead, it is likely that aggressive behaviour following consumption is a result of the disruption of cognitive mechanisms closely associated with the behaviour (Attwood & Munafo, 2014) that in turn increase the likelihood of aggressive responding (Bushman, 1997). Several well-established theories have been proposed to explain the causes and manifestation of aggressive behaviour. Most noteworthy, are the Cognitive Neoassociation (Berkowitz, 1989, 1990), Social Learning (Bandura, 1973, 2001), and Social Interaction (Tedeschi & Felson, 1994) theories amongst others (for review, Geen & Donnerstein, 1983).

1.2.3. Theoretical and Meta-Theoretical Framework of Aggression

The Cognitive Neoassociation model builds upon the earlier Frustration-Aggression Hypothesis (Berkowitz, 1989; Dollard et al., 1939) and according to this framework negative affective states influence aggressive behaviour. More specifically, aversive events such as frustration, provocation and receiving physical pain can result in affective, cognitive, motor responses most associated with aggression (Slotter & Finkel, 2011). In the context of alcohol consumption, this theory may explain why perceived frustration and provocation may lead to

aggressive behaviour whilst intoxicated, as both frustration and provocation have been linked to increased aggressive responding (Giancola et al., 2002; Gustafson, 1985). According to Social Learning Theory, individuals respond aggressively after observing other individuals behaving aggressively (Bandura, 1973). Given that alcohol is often consumed socially, it is reasonable to speculate that observed alcohol related aggression within a social context is likely to influence the drinkers own behaviour according to social learning theory. Finally, Social Interaction Theory suggests that aggressive behaviour is goal driven. For example, individuals act aggressively within a social environment to obtain something of value, overcome or surmount threat, exact retribution, or establish a desired social identity (Tedeschi & Felson, 1994). These theories individually offer different explanations surrounding the likelihood of an individual acting aggressively.

The I^3 theory, meta-theoretical model of aggression that builds upon the General Aggression Model (GAD) proposed by Anderson and Bushman (2002), was introduced to provide a framework for understanding human aggression that encompasses previous models of aggressive behaviour (Slotter & Finkel, 2011). There are three main components of this framework that each increase the likelihood of an individual responding aggressively, and these behaviours can be predicted based on the strength of *Instigation,* degree of *Impellence* and control of *Inhibitory* factors (Parrott & Eckhardt, 2018). Instigating triggers such as provocation have been shown to increase aggressive behaviours (Berkowitz, 1993), and according to the I^3 framework, these triggers function as the initial momentum towards the behaviour. Impelling factors are individual and/or situational factors that increase the individual's propensity towards aggressing. For example, individuals with high trait anger are more susceptible to aggression (Birkley & Eckhardt, 2015), and are therefore more likely to respond aggressively to cues of provocation and other instigating factors. The final component of this framework is inhibitory control. According to this, inhibitory

factors increase the likelihood that an individual can resist the urge to respond aggressively in response to an instigating trigger and despite the individual propensity to react in a particular way (Parrott & Eckhardt, 2018). In the context of alcohol consumption, the I^3 model provides a framework for addressing why some, but not all individuals respond aggressively when intoxicated (Finkel et al., 2012) or as a result of long-term exposure to the substance. An example of this application comes from a laboratory study that found that individuals with high trait anger (an impelling factor) was associated with greater aggression to provocation (a strong instigating factor), and this was only found in intoxicated men (inhibitory control impaired) (Parrott & Giancola, 2004). This framework can also be used to understand other potential contributing cognitive mechanisms of alcohol related aggressive behaviours. Explanations include the impairing effects of alcohol consumption on executive function and behavioural control (i.e., disinhibition of behaviour) (Abroms et al., 2003; Field et al., 2010), stress-dampening (i.e., reduced anxiety and increased approach tendencies) (Sayette, 1993), and the perception of socially relevant cues associated with aggression (i.e., erroneous perception of provocation and threat) (Pernanen, 1991; Steele & Southwick, 1985). The role of these socially relevant cues is especially important given that alcohol is often consumed within a social context.

1.2.4. Cognitive Mechanisms of Alcohol Related Aggression

1.2.4.1. Disinhibition of Behaviour

Executive functioning is a series of cognitive abilities that collectively control behaviour (Suchy, 2009). Evidence reliably suggests that alcohol consumption impairs cognitive functioning (Bartholow et al., 2003; Casbon et al., 2003), and more specifically, inhibitory control (Giancola, 2000); a core component of executive functioning alongside working memory and mental flexibility. Poor inhibitory control has also been shown to be a factor contributing to impulsive

responding (de Wit, 2009) and is therefore key in the avoidance of maladaptive behaviour and problematic behaviour. Several experimental studies have reliably demonstrated inhibitory control impairment following a moderate dose (0.4-0.45g/kg) of alcohol (de Wit et al., 2000; Marczinski et al., 2005). These processes are linked to prefrontal regions of the brain, which have been shown to be disrupted following acute alcohol consumption and prolonged chronic consumption (Harris et al., 2008; Makris et al., 2008; Oscar-Berman & Marinkovic, 2007). It is therefore likely that socially unacceptable behaviours and aggressive responding may be influenced by the disinhibiting effects of alcohol. Support for this comes from research investigating clinical populations with response inhibition deficits (i.e., ADHD) (Puiu et al., 2018). Poor inhibitory control and impulsivity in these clinical samples is related to aggression (Pawliczek et al., 2013; Raaijmakers et al., 2008). Similarly, research has also shown that better executive functioning (Hoaken et al., 2003) and specifically better self-control (i.e., the ability to inhibit maladaptive behaviours) (Denson et al., 2011; DeWall et al., 2007) is inversely linked to aggressive behaviour. It is clear that impulse control and response inhibition are important mechanisms involved in aggressive behaviour, and alcohol induced impairment of these processes may function to increase the likelihood of these behaviours occurring.

1.2.4.2. Stress Dampening

Another indirect mechanism by which alcohol may increase aggression is via its anti-anxiolytic properties (Sayette, 1993). These authors proposed the appraisal distribution model which suggests that stress dampening as a result of alcohol consumptions occurs because the appraisal of stressful information is disrupted. This results in lower negative affect and anxiety in relation to social sanctions and personal harm. This was supported by experimental research that tested whether alcohol consumption would reduce stress when consumed prior to the exposure of

a stressor (stress induced by asking participants to present a speech about their own appearance) (Sayette et al., 2001). They found that alcohol tended to attenuate stress responses when the exposure to the stressor followed consumption. They concluded that alcohol was more likely to reduce stress when the appraisal occurred during intoxication, in line with the appraisal distribution model. The likelihood of aggressive responding may therefore be increased due to alcohol induced anxiety and stress reduction, paired with the reduced tendency to relent when threatened, and/or increased likelihood to approach threatening or hostile situations (Sripada et al., 2011; Vogel-Sprott, 1967) .

1.2.4.3. Alcohol Myopia

Another prominent concept used to explain alcohol related aggressive behaviour is Alcohol Myopia Theory (Steele & Josephs, 1990). This theory suggests that alcohol intoxication impairs cognitive processes and influences aggressive behaviour by narrowing attentional focus. It creates a myopic effect in which attention can only focus on the most salient and easily processed cues within a social environment. Giancola et al. (2011) describe that in hostile situations the myopic effect of alcohol increases the likelihood of violence by narrowing attention on cues of potential provocation. Since these cues are perceived to be threatening, they are particularly salient and therefore more attentional resource would be allocated, whilst non-threatening/provocation cues are assigned limited attentional resource and are less likely to be processed. This explanation has been used to explain several maladaptive and problematic behaviours following alcohol consumption, such as risky sex behaviour (MacDonald et al., 2000) and drink driving (Giancola et al., 2010). Additional support comes from alcohol laboratory studies that distract individuals from provocation and threatening cues. These studies highlight that when attention is shifted to inhibitory cues that attenuate aggression (i.e., non-threatening), physical aggression is reduced in

men (Gallagher & Parrott, 2011; Giancola & Corman, 2007). Cross-sectional evidence suggests that individuals with a predisposition towards aggression related cognitive biases (Leonard & Blane, 1992) are highly susceptible to shifts towards cues of provocation following alcohol consumption (Gallagher et al., 2010) and, as a result, are typically aggressive when intoxicated. Similarly, past research highlights that individuals with aggressive tendencies have an attentional bias towards aggressive cues (Eckhardt & Cohen, 1997; Smith & Waterman, 2004). It therefore reasonable to speculate that the myopic effect of alcohol may actually attenuate aggression amongst individuals that are not typically aggressive, or do not have aggressive traits, as the most salient cues to these individuals might not be those that threaten or provoke.

There is support for this counterintuitive prediction that alcohol consumption can actually have the opposite myopic effect and actually reduce aggression in certain types of individual (Gallagher & Parrott, 2016; Purvis et al., 2016). These authors conclude that individual differences in the susceptibility to the myopic effects of alcohol, influence the attentional focus and cue salience when intoxicated. These findings are of particular interest as they have implications for interventions aimed at reducing alcohol related aggression (Giancola, Josephs, et al., 2009) and highlight the need to consider differences in aggressive personality traits. The Alcohol Myopia Theory describes an effect that is an important inhibitory factor outlined in the aforementioned I^3 model of aggression (Parrott & Eckhardt, 2018; Slotter & Finkel, 2011). This theory should therefore be considered in parallel with the I^3 model to allow a more holistic focus on instigating factors (i.e., cues of threat and provocation), impelling forces (i.e., individual and situation differences in aggressive tendencies), as well as inhibitory control (i.e., behavioural control and the myopic effect on attentional focus). It can therefore be argued that Alcohol Myopia Theory and the I^3 Model of aggressive behaviour taken together provides a framework for the role of social

cues and individual factors on alcohol induced aggressive behaviour, given that alcohol is most typically consumed socially. There is clear support for multiple cognitive mechanisms by which alcohol could increase the likelihood of aggressive behaviour. A relatively overlooked mechanism that may also contribute to the increased likelihood of aggressive behaviour following alcohol is emotional face processing (Attwood & Munafo, 2014).

1.3. Alcohol and Emotional Face Processing

1.3.1. Emotional Face Processing

Emotional facial expressions are important social cues and non-verbal forms of communication that are considered to be a fundamental component of effective social interactions (Moriya et al., 2013). Ekman (1992) described six universally recognised emotional facial expressions (i.e., anger, sadness, happiness, disgust, fear, surprise) as a rich source of social information that allow the perceiver to infer thoughts, feelings, moods and intentions of others, and that are capable of influencing behaviour (Eisenberg et al., 1989; Klinnert, 1983; Marsh et al., 2007). The ability to recognise displays of emotion in faces develops at a very early age, as young infants use these displays as behavioural cues (Mancini et al., 2018). In fact, evidence suggests that children as young as 12 months old can recognise emotion and adjust their behaviour accordingly during a social interaction with a caregiver (Hertenstein & Campos, 2004). In childhood, processing ability appears to differentially develop depending on the emotional expression. For example, children are capable of processing happy and sad facial expression from a young age, with a poorer ability to recognise anger and disgust (Gosselin, 1995). These authors conclude that positive emotion perception develops earlier than negative. This preference for positive over negative emotional expressions shifts in adulthood as negative emotions are attended to for longer periods and can be used to guide behaviour (Klinnert, 1983). The ability to process

emotional facial expressions in childhood has an impact on later academic competence and the ability to form social relationships (Izard et al., 2001). Research also highlights that children with high aggressive personality traits typically demonstrate some form of emotional face processing impairment (Kimonis et al., 2006). The ability to process emotional facial expression typically develops and improves as age increases (Theurel et al., 2016), and the emergence of matured recognition patterns occur in early adulthood (Herba & Phillips, 2004).

In adults, sad and fearful facial expressions are distress cues that promote prosocial behaviour in others and inhibit aggression (Eisenberg et al., 1989; Marsh et al., 2007), whilst angry expressions may reduce socially unacceptable behaviour in some individuals (Blair et al., 1999). However, approach behaviours have been reported if anger expressions are perceived as threatening, and if the threat is considered surmountable (Wilkowski & Meier, 2010). Deficits in the ability to recognise emotion in facial expressions is associated with poorer social function (Blair, 2003). For example, failure to process distress cues (i.e., sadness and fear) (Blair, 2005) and misidentification of anger (Hall, 2006) have been associated with inappropriate aggressive responding tendencies. Emotion recognition has been linked to key areas of the brain responsible for processing this information (Adolphs, 2002; Barbas et al., 2003; Davidson et al., 2000). Neuroimaging research has described key areas of the brain that are responsible for emotional face processing (Henderson et al., 2014). It is likely that alcohol impairs neural function associated with emotion processing. Similarly, evidence demonstrates that clinical and forensic samples display emotion processing deficits (Demenescu et al., 2010; Hoaken et al., 2007; Marwick & Hall, 2008). This evidence is also reviewed, and it is argued that alcohol consumption results in a similar profile of impairment displayed in these populations.

1.3.1.1. Neurological Framework of Emotion Recognition

Adolphs (2002) describe several key areas of the brain that are responsible for the processing of emotion displayed in a facial expression. These areas include the amygdala, orbitofrontal cortex, and anterior cingulate cortex, which are highly interconnected regions. Evidence has suggested that behavioural changes can be attributed to damage to these brain regions (Barbas et al., 2003; Davidson et al., 2000). There is some debate in the literature surrounding the function of the amygdala in the processing of emotional facial stimuli. Some evidence suggests that emotions that are most associated with behavioural withdrawal are more dependent on amygdala activity (Anderson et al., 2000), however most evidence tends to argue that the amygdala is primarily involved in the processing of emotional facial stimuli most closely linked to threat and danger (Adolphs & Tranel, 2000; Adolphs et al., 1999; Ohman, 2005; Ohman & Mineka, 2001). The latter is supported by studies that report greater amygdala activation in response to processing fear evoking stimuli (Romanski & LeDoux, 1992) and fearful facial expressions in functional imaging studies (Breiter et al., 1996; Morris et al., 1996). Similarly, research has shown bilateral damage to the amygdala results in impaired processing of fearful expressions (Adolphs et al., 1999). Some support for the former (i.e., the amygdala being responsible for processing emotions associated with withdrawal) comes from research identifying the role it plays in the processing of sad emotions (Wang et al., 2005). However, this has not been consistently reported (Blair et al., 1999).

The prefrontal cortex (PFC) activation has been shown to be involved in the experience of anger and expressive aggression (Rule et al., 2002). Neurological evidence also suggests that specific sub-regions of the PFC, in particular, the orbitofrontal cortex (OFC) are involved in face processing (Coccaro et al., 2007). Evidence to support these claims suggests that damage to the

OFC, especially the right, has been found to impair the recognition of facial expressions (Hornak et al., 1996). More specifically, selective activation in the right OFC was demonstrated in response to angry faces compared to other negative emotions (Blair et al., 1999). Similarly, a study utilising transcranial magnetic stimulation to disrupt the medial prefrontal cortex found that participants produced longer reaction times in response to angry but not in response to happy faces (Harmer et al., 2001).

1.3.1.2. Emotional Face Processing in Clinical and Forensic Samples

Since effective processing of emotional facial expressions is a fundamental component of a successful social interaction, it is reasonable to speculate that a breakdown in emotional and social responding may be in part a result of a processing impairment (Gillespie et al., 2015) The impaired ability to process emotional facial expressions has been reported in a number of different clinical disorders (Demenescu et al., 2010; Marwick & Hall, 2008). Demenescu et al. (2010) reviewed 18 studies exploring explicit emotion recognition in patients with major depression and anxiety disorder compared to healthy controls. They report that adults with major depression (d=-0.58) or anxiety disorders (d=-0.35) were significantly poorer at recognising emotions. Further, research has shown that socially anxious individuals tend to misidentify neutral facial expressions as angry (Mohlman et al., 2007) and this is supported by similar research that reports a greater sensitivity towards negative emotions (Joormann & Gotlib, 2006). There is also evidence to suggest that depressed patients have a tendency to identify happy faces as neutral (Leppanen et al., 2004). This has implications for effective social functioning as the reduced tendency to see positive emotions paired with the propensity to see threat or cues of provocation (i.e., angry faces) may increase the likelihood of maladaptive responding. Other clinical cohort studies involving schizophrenia patients show impaired emotion processing deficits, especially in individuals that

present psychopathic or violent traits (Marwick & Hall, 2008). This is best supported by Blair (2005) who proposed an integrated model of psychopathy and cognition. This model highlights that psychopathic tendencies result in failure to process threatening cues (i.e., sadness and fear) which can lead to aggressive responding.

The relationship between deficits in emotional face processing and aggressive behaviour is probably best supported by research investigating typically aggressive populations including violent offenders (Hoaken et al., 2007), sex offenders (Gery et al., 2009), autistic individuals with attention deficit hyperactivity disorder (Sinzig et al., 2009), patients with intermittent explosive disorder (Best et al., 2002), and individuals with psychotic traits (Blair et al., 2001). A review compared emotion recognition in violent vs non-violent offenders and reported that violent offenders were less accurate at identifying negative emotions compared to non-violent offenders. Specifically, the most reported impairment amongst the studies reviewed were deficits in disgust identification accuracy (Robinson et al., 2012; Seidel et al., 2013) and a reduced sensitivity to processing fearful faces (Gillespie et al., 2015; Schonenberg et al., 2014; Schonenberg et al., 2013). The lower accuracy in disgust recognition is consistent with a study that highlights the same impairment in recognising disgusted faces amongst a sample of psychopathic prison inmates (Kosson et al., 2002). In support of these findings, Penton-Voak et al. (2013) found that self-reported anger and aggression could be improved by promoting happiness recognition over anger. This study involved training a sample of healthy individuals as well as a group of adolescents with behavioural problems to recognise happiness more frequently. Results indicate that both groups demonstrated a shift in bias towards happy faces, and this led to lower self-reported anger and aggression. These findings from clinical and forensic populations, as well as emotion recognition training, highlight the potential importance of emotion recognition as a key influential factor of

aggressive behaviour. As alcohol consumption is linked to a causal increase in aggression, it is plausible that emotion processing impairment may be one mechanism in which the likelihood of this behaviour is increased. Therefore, it is anticipated that alcohol consumption may induce similar emotion processing deficits which will increase the likelihood of aggressive behaviour.

1.3.2. Acute and Chronic Alcohol Consumption and Emotion Processing Deficits

In most western countries, alcohol is often consumed socially and is expected to improve interpersonal communication, sociability and enjoyment by promoting positive and attenuating negative emotions (Capito et al., 2017). It is widely accepted that it is often exploited as a form of social lubricant aimed at improving social interactions, and this is considered to be one of the main motivations for consumption (Cooper et al., 1995; Cox & Klinger, 1990; Goldman et al., 1987; Miller et al., 2015; Monahan & Lannutti, 2000). Given the social context alcohol is often consumed in, the processing of emotional facial expressions is considered to be an important factor in the success of an interaction. Conduct disorder research suggests that the propensity to respond aggressively and in a maladaptive manner may be due to a combination of impairment of both ability to recognise emotional stimuli and the cognitive control of emotional behaviour (Sterzer et al., 2005). Attwood and Munafo (2014) argue that a similar impairment profile may exist following the consumption of alcohol resulting in a similar propensity to respond aggressively. For example, impaired processing of socially relevant facial cues that function to promote sociability (i.e., happy faces), an increased tendency to see provocation signals (i.e., angry faces), both paired with reduced inhibitory control may increase the likelihood of aggressive responding when intoxicated. Recent reviews indicate that both chronic (Donadon & Osorio Fde, 2014) and acute alcohol consumption (Attwood & Munafo, 2014) can alter the processing of emotional facial expressions.

1.3.2.1. Chronic Alcohol Consumption

Alcohol dependence and alcoholism has been linked to long term deficits in neurological functioning, including learning, memory, visuo-spatial orientation, information processing, amongst other (Bates et al., 2002). Research has also shown that this excessive chronic consumption produces pronounced impairment in the abuser's ability to process and recognise emotional and/or affective information (Kornreich et al., 2001; Maurage, Campanella, Philippot, Martin, et al., 2008). As a result, these impairments promote deficits in social cognition, and impact the ability to interact and adapt within a social environment (O'Daly et al., 2012; Uekermann & Daum, 2008). Deficits in emotional face processing have been documented in alcohol abusers (Foisy et al., 2007; Townshend & Duka, 2003). Specifically, Donadon and Osorio (2017) demonstrated that individuals who are alcohol dependent are poorer at accurately identifying emotions, require greater emotion intensity and take longer to make accurate judgements, compared to healthy controls. Specifically, emotion processing accuracy impairment was reported to be poorer amongst alcoholic patients compared to healthy controls in several studies (Foisy et al., 2007; Kornreich et al., 2003; Kornreich et al., 2002; Maurage, Campanella, et al., 2007). Research also demonstrated that sadness and disgust were more poorly recognised by alcoholic groups in comparison to healthy controls, and that these groups have a tendency to erroneously identify emotions such as sadness as anger when processing facial expressions, suggesting an anger perception bias (Frigerio et al., 2002; Philippot et al., 1999). What remains unclear is whether chronic alcohol consumption within the general population (i.e., non-dependent samples) is associated with poorer emotion recognition. It is likely that frequent and habitual drinking patterns in regular, social drinkers may mimic emotion processing deficits seen in alcohol dependent participants but to a lesser extent.

The exact nature of the impairment is unknown but a plausible explanation that provides insight into why alcoholism may be associated with emotion processing deficits surrounds the degeneration of specific brain region activation due to prolonged heavy drinking. Research using electroencephalogram (EEG) measured brain activity whilst alcoholic patients completed cognitive tasks. This evidence suggests that lower activation in brain areas associated with visual motor processing was exhibited by alcoholics, and that this may mediate deficits in angry face processing (Maurage, Campanella, Philippot, de Timary, et al., 2008; Maurage, Campanella, Philippot, Vermeulen, et al., 2008; Maurage, Philippot, et al., 2008; Maurage, Philippot, et al., 2007). Magnetic resonance imaging (MRI) studies have indicated that alcoholics show low activation in the orbitofrontal, cingulate and insular cortex during the recognition of disgusted (O'Daly et al., 2012) and fearful faces (Salloum et al., 2007). Evidence also suggests that alcoholics exhibit lower right amygdala and hippocampus activation when viewing both positive and negative emotional stimuli in comparison with non-alcoholic controls (Marinkovic et al., 2009). It is clear that prolonged alcohol consumption produces deficient activation of several brain areas responsible for emotion regulation and processing (Donadon & Osorio, 2017). Contrastingly, there is less research exploring the acute effects of alcohol on emotional face processing among non-dependent/problematic samples (i.e., typical social drinkers). It is likely that the effects within a single drinking session are different to the impairment reported on alcoholic individuals.

1.3.2.2. Acute Effects of Alcohol Consumption

Early work exploring acute alcohol consumption and emotion processing reports that overall accuracy in recognising facial expressions was impaired by consumption, and this effect was enhanced by the expectation of receiving alcohol (Tucker & Vuchinich, 1983). These authors used a balanced placebo design in which participants received either alcohol (0.5g/kg) or a placebo

and were told to expect or not expect alcohol. Analyses were not stratified by emotion, so the emotion specific deficits of alcohol were not tested. In other research emotion specific effects have been investigated, but outcomes are inconsistent. Research investigating the prosocial effects of alcohol on emotion processing report a reduction in the time taken to recognise happy faces following acute consumption (Kano et al., 2003). However, these effects were only present at relatively low doses of alcohol (0.14g/kg). Furthermore, as the dependent measure was reaction time, not response categorisation (i.e., deciding what the emotional expression is), the mechanism by which alcohol impacts emotion perception is difficult to determine. Dolder et al. (2017) similarly report that alcohol results in faster recognition of happy facial emotion. These authors measured emotion perception accuracy (i.e., categorisation) and intensity (i.e., threshold percentage of emotional intensity required to make an accurate judgement). The results from this study indicate that low doses of alcohol do not affect recognition accuracy of any emotional expression, but it does influence the speed in which happy expressions are recognised (i.e., lower percentage of happiness required to accurately identify the emotion) compared to placebo. It was argued that these findings highlight a greater propensity to see happy emotions following alcohol which is likely to promote sociability. Taken together these studies indicate that low doses of alcohol seem to increase the ability to process happy expressions.

Other research suggests that acute alcohol consumption alters processing of negative emotional expressions, which in turn could influence alcohol related aggression (Attwood & Munafo, 2014). For example, a decreased sensitivity towards perceiving sadness has been reported following acute alcohol consumption (Attwood, Ohlson, et al., 2009; Craig et al., 2009). These studies used two-alternative forced choice (2AFC) tasks to test discriminatory thresholds. Emotional expression continua were produced for angry, sad and happy faces each ranging from

prototypical neutral to full emotional expression. Results suggested that alcohol modifies the perceptual threshold for sad facial expressions, indicative of reduced ability in recognising sadness (i.e., reduced sensitivity). This has implications for alcohol-related aggression, as sadness is an indicator of submission (Hart, 2011), which may curtail aggression. In turn, reduced perception of sadness could therefore increase aggression by negating an important inhibitory signal that would reduce likelihood of aggression. Similar to evidence that argues alcohol increases the speed of happy recognition (Dolder et al., 2017; Kano et al., 2003), these authors focus on perceptual thresholds rather than expression categorisation. Whilst this approach allows authors to test which happy face is the happiest and which angry face is the angriest, it does not test the exact mechanism influencing the threshold change nor does it allow the misattribution of one emotion for another to be explored.

An increased bias towards perceiving angry faces (in ambiguous negative facial morphs) has been reported following acute alcohol consumption (Attwood, Ataya, et al., 2009). This study used a similar 2AFC task presenting angry-happy and angry-disgust morph sequence. Full exemplar of the expressive face was presented at each end of the continuum (i.e., full angry expression to full happy/disgust expression) and ambiguous in the middle (i.e., a blend of the two target emotions). Balance-point scores were used to measure a tendency to perceive one emotion over the other in each 2AFC emotion pair; a high score would indicate a tendency to see angry whilst a low score indicates a tendency to see happy/disgust. Participants demonstrated a greater tendency to see anger in male expression on the angry-disgust emotional continuum following alcohol consumption compared to placebo. In contrast, there was no evidence of an effect of alcohol on the angry-happy morph sequence. This altered processing of angry facial expressions is likely to have a meaningful impact on behaviour, as a bias towards seeing anger may increase

perceived provocation, which is a primary driver of aggression (Giancola et al., 2002). More recent studies have measured the effects of acute alcohol consumption using tasks presenting the six basic emotions (anger, sadness, happiness, disgust, fear, surprise). Felisberti and Terry (2015) investigated emotion recognition of the six basic emotions following a low (0.17 g/kg females; 0.20 g/kg males) and high (0.52 g/kg females; 0.60 g/kg males) doses of alcohol. They report that the recognition of disgust and contempt emotions was better following high doses. Similar research investigated the influence of alcohol on the processing of dynamic emotional stimuli (anger, sadness, happiness, disgust, fear, neutral). They report a tendency to misclassify sad emotion as neutral lending partial support to the conclusions drawn by Attwood, Ohlson, et al. (2009) and Craig et al. (2009) who similarly report a reduced sensitivity towards sadness. In addition, Craig et al. (2009) highlight the importance of utilising prototypical facial expression stimuli (i.e., composite images generated from multiple individuals) that reduce idiosyncratic differences between individuals. More recent research using composite images of the six basic emotions found weak evidence supporting an anger bias after alcohol consumption, but effect sizes are small (Khouja et al., 2019). It is clear that there is no consensus for how acute alcohol influences emotional face processing. The use of different emotion recognition and discrimination tasks in previous research may contribute to the inconsistent conclusions and makes comparing outcomes difficult. There is some evidence that explores the interpretation of emotional facial expressions, specifically whether greater hostility is perceived, rather than the categorisation of the displayed emotion (Smeijers et al., 2017). The focus on interpretation and evaluation may be key in understanding how emotional facial expressions mediate the alcohol-aggression relationship.

1.3.3. Hostile Attribution Bias of Emotional Facial Expressions

The tendency to perceive or interpret others' behaviour as hostile is often referred to as hostile attribution bias (Nasby et al., 1980). Research suggests that higher levels of this bias are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006). This can have negative social consequences, as perceived aggressive intent plays a causal role in reactive aggressive behaviour (Crick & Dodge, 1996). Within the literature, an increased bias towards seeing anger has been interpreted as an increased bias towards judging facial expressions as hostile (Wegrzyn et al., 2017). However, 'anger' and 'hostility' conceptually differ (Eckhardt et al., 2004). Anger is an emotion most associated with feelings of irritation, annoyance, fury and rage. State-anger is often described as the response to an emotional elicitor that induces these feelings, whilst trait-anger is considered to be a more constant personality trait characterised by more frequent experiences of these feelings even when the cues are innocuous or unprovocative (Ramírez & Andreu, 2006). Hostility on the other hand, can be considered to be an individual attitude that involves negative evaluations of others (Eckhardt et al., 2004), and therefore may be better indicator of intentions of an individual. The perception of hostility communicates the intention to harm an individual, including expressive characteristics that signal intent for physical violence (Deffenbacher, 2000). In support of a difference between anger and hostility, one study found that facial displays of hostility produced greater physiological arousal than displays of anger (Tsikandilakis et al., 2020).

Hostile interpretations may not be restricted to angry faces. It is likely that other emotions, or emotionally ambiguous facial expressions, may also be interpreted as hostile; a disgusted face in particular may be judged as more hostile as it shares similar expressive characteristics to anger (Wieser & Brosch, 2012). Recent research has investigated hostile attribution bias in facial affect

using a sample of typically aggressive individuals (i.e., forensic outpatient population) (Smeijers et al., 2017). This research presented individuals with images of four facial expressions of emotion (angry, fear, disgust, and happy) which were judged as either displaying hostility or not. They found that individuals with an aggression regulation deficit (i.e., antisocial and borderline personality disorder) demonstrate an increased perception of hostility in emotional expressions (angry, disgusted, fearful and happy faces) compared to healthy controls. The authors discuss this hostile attribution bias towards emotional stimuli as a key characteristic of pathological aggression in a forensic outpatient sample. This appears to be the first study to investigate hostile attribution bias of emotional stimuli by treating angry expressions and hostile judgements as separate concepts. What is not clear is whether a similar profile of hostility is perceived in emotional facial expressions following alcohol consumption. The evidence reviewed highlights the importance of isolated facial expression as key social information. Alcohol consumption is anticipated to influence the way in which emotions are recognised and interpreted. More recent research has started to expand our understanding of the social world by exploring inferences made when viewing social interactions (i.e., others interacting). The way in which this information is processed and judged during an encounter is likely to be influenced by alcohol consumption.

1.3.4. Dyadic Social Interactions Perception

Understanding the way in which impressions of others are formed, and how others form impressions of us is important for determining the success of social interactions (Quadflieg & Penton-Voak, 2017). Typically, past literature has focused on the perception and evaluation of isolated individuals (Ko, 2018). This research has shown that emotional expressions of individual facial expressions are a fundamental component of effective social interaction (Moriya et al., 2013). These expressions inform the perceiver of the expressors' emotional state and behavioural intent

(Eisenberg et al., 1989; Klinnert, 1983; Marsh et al., 2007). Evidence also suggests that the perception of another person's body shape/posture and direction of movement can also influence impressions formed about their intentions, personality traits and emotional state (de Gelder, 2006; Macrae & Quadflieg, 2010; Uleman & Saribay, 2012). Emerging evidence suggests that impression formation when observing others interacting may directly influence the observer's behaviour. Quadflieg and Westmoreland (2019) argue that forming impressions when observing third party encounters (i.e., dyadic social interactions) can directly influence the perceivers own behaviour and intentions towards others, even if the impressions formed are inaccurate.

Individuals tend to be better at judging the type of relationship between two people interacting than the quality of the relationship. For example, most people are accurate at judging whether two people know each other, or are romantically involved (Latif et al., 2014; Place et al., 2009), but struggle to judge the degree of rapport or how much they like each other (Bernieri & Gillis, 1995; Bernieri et al., 1996; Floyd & Erbert, 2003). This has important implications in relation to aggression, as the ability to distinguish between two people that know each other behaving aggressively (e.g., play fighting), and two people who do not know each other behaving aggressively (e.g., violent altercation) will differentially influence the perceivers behaviour and attitudes towards that interaction. Observations of third party encounters can also inform the perceiver of potential threat with regards to their own personal social standing (Mast & Hall, 2004), and this in turn can influence tendency to approach or avoid a particular situation (Milinski, 2016). For example, observers can identify individuals prone to hostile, unsociable and potentially dangerous behaviour (Hamlin, 2013).Therefore, perceived positive (e.g., caring, protection, co-operation) and negative (e.g., hostility, dangerous, unfair, volatile) interactions have the ability to

influence the likelihood of the perceiver to interact with those who they observe (Quadflieg & Westmoreland, 2019)

As previously reviewed, it has been shown that acute alcohol consumption impairs the processing of emotional facial expressions when perceiving isolated individuals (Attwood & Munafo, 2014). What remains unclear is whether impressions/interpretations formed from viewing dyadic social interactions (i.e., observations of two or more individuals interacting) are influenced by acute alcohol consumption. As these interactions provide important social insights, disruption or impairment to the perception of these can potentially influence behaviour. It is likely that a similar profile of impairment is apparent when individuals perceive isolated facial expressions and a dyadic social interaction. In the context of aggression, alcohol could result in the perception of greater hostility perception when seeing ambiguous or benign social interactions which may increase the likelihood of aggressive responding. It is apparent that emotional expressions as well as dyadic social information are important when considering aggressive behaviour. It is anticipated that alcohol consumption plays a functional role in how this information is processed and interpreted. It is also worth considering individual differences that may contribute to a person being more susceptible to aggression when intoxicated. Specifically, differences in gender, outcome expectations, personality types that make an individual more susceptible to perceptual biases.

1.4. Individual Differences in Aggressive Behaviour

As previously mentioned, not everyone that consumes alcohol becomes aggressive (Bushman & Cooper, 1990). Given that only a small proportion of consumers reliably display alcohol related aggression, it is reasonable that individual differences may contribute to aggression in some but not all drinkers. Understanding these differences is important to fully understand the

mechanisms involved in increasing the likelihood of aggressive responding, and for the development of intervention (Parrott et al., 2012). It is also likely that these individual differences influence the perception of emotional facial expressions. Whilst these individual differences are reviewed independently of one another below, it is typically assumed that these factors interlink and interact.

1.4.1. Gender

Evidence generally shows that aggressive traits are more prominent in men (Archer, 2004; Quinn et al., 2013). In a meta-analysis conducted by Archer (2004), males demonstrated more direct and frequent physical aggression when compared to females. This author argued that this gender difference may be influenced by masculine expectations (i.e., agentic traits in males) resulting in males learning that aggressive responding is appropriate, as well as early socialisation during childhood encouraging boys to play more competitively and aggressively. Experimental evidence to support these conclusions found that the acute administration of alcohol increased aggression in males compared to female participants (Giancola, Levinson, et al., 2009). Biological mechanisms have been proposed to explain gender differences in alcohol-related aggression including differences in sex hormones associated with aggression (Archer, 2006; Oyegbile & Marler, 2006; Soma, 2006). In particular, a single dose administration of testosterone (0.5mg) induced an accelerative cardia response to angry faces in female participants (van Honk et al., 2001), and testosterone was also found to be associated with increased approach to and reduced avoidance of angry facial expressions signalling threat (Wirth & Schultheiss, 2007). Evidence also highlights a positive correlation between testosterone and amygdala activity in response to fearful and angry facial expressions (Derntl et al., 2009).

Although evidence suggests that males are typically more aggressive following alcohol, there is emerging research that argues, in more recent times, that females act in more "masculine" ways resulting in more aggressive behaviour. Newberry et al. (2013) discuss that the frequency of binge-drinking among women has increased and found that the perpetration of an aggressive act, particularly verbal aggression in response to a grievance, was associated with an increase in alcohol consumption. Similar research indicates that both males and females were more likely to use verbal and physical forms of aggression when drinking, and for females in particular, drinking was associated with relational aggression and anger (Robertson et al., 2020). It is clear that whilst the general consensus is that aggression (especially physical displays) is more prominent among males, it is not an exclusive male phenomenon. Aggressive personality traits (i.e., high trait aggression) in both males and females increases the likelihood of aggression (Giancola, 2002b), and therefore individual differences in personality type may be another potential mediator of alcohol induced aggression. Giancola, Levinson, et al. (2009) also experimentally tested the acute effects of alcohol on aggression in men and women using a modified version of the Taylor Aggression Paradigm. They report that both men and women were more aggressive following alcohol, and the effect was more pronounced in men. Again, this evidence supports the claim that both men and women respond more aggressively when intoxicated and also supports the argument that men display greater alcohol induced aggression.

1.4.2. Expectancies

Alcohol outcome expectancies often develop through direct experience (i.e., previous drinking occasions), through observations and/or from cultural and social norms (Blume & Guttu, 2015). It is commonly believed that alcohol increases aggression, although this belief normally centres around others becoming aggressive when drunk rather than the individual themself

believing they will become aggressive (Paglia & Room, 2006). Expecting alcohol to increase aggression has been shown to mediate alcohol related aggression (Dermen & George, 1989). These authors found that the relationship between drinking habits and physical aggression was stronger when participants expected alcohol to increase aggression when compared to having no expectation or expecting a decrease. Similar research has also shown that expectancies mediate the relationship between both verbal and physical intimate partner aggression in men and women and excessive drinking, again highlighting the key role expectation plays in aggressive behaviour. Experimental work shows that individuals who believe they have consumed alcohol also respond more aggressively even when they haven't received alcohol (i.e., placebo condition) (Bushman, 1997). It is clear from this that the expectation of alcohol alone can alter behaviour and emphasises that aggressive responding is not purely pharmacological. Instead, it is likely that alcohol consumption triggers aggression outcome expectancies and these expectations increase the likelihood of the behaviour. It's therefore plausible that changing alcohol outcome beliefs can contribute to reducing aggressive responding.

1.4.3. Personality Traits

Individual differences in personality traits have been theorised to influence the alcohol-aggression relationship (Chermack & Giancola, 1997); alcohol facilitates aggressive behaviour among individuals who are predisposed to act aggressively when in a sober state (Collins et al., 1988; Pernanen, 1991). Research has highlighted that intoxicated individuals with high or moderate levels of trait anger demonstrated greater physical aggression when compared to intoxicated individuals with low levels of trait anger (Parrott & Zeichner, 2002). Of note, studies have shown that higher levels of trait anger (Parrott & Zeichner, 2002), irritability (Giancola, 2002c) and trait aggressiveness (Giancola, 2002a) influence alcohol related aggression. In

addition, anger control (Parrott & Giancola, 2004) and lower levels of dispositional empathy (Giancola, 2003) has also been shown to potentiate aggressive behaviour following alcohol consumption. Of these personality traits, trait aggressiveness reflects an individual predisposition to respond aggressively (Buss & Perry, 1992) and has gained a lot of research attention surrounding its role as a potential risk factor involved in increasing aggressive behaviour. Acute alcohol consumption has consistently been shown to increase aggression among individuals who report higher levels of trait aggression in comparison to those who report low (Eckhardt & Crane, 2008; Giancola, 2002a; Giancola et al., 2005; Moeller et al., 1998).

These personality traits have also been shown to influence emotional facial expression processing in sober individuals. Individual differences in trait anger and approach motivation tendencies have been linked to greater amygdala activity when perceiving angry facial expressions (Beaver et al., 2008). In addition, individuals with impulsive aggression traits demonstrate amygdala hyper-reactivity to expressions of angry expressions (Coccaro et al., 2007). Since the amygdala is responsible for processing signals of threat and danger (Adolphs & Tranel, 2000; Adolphs et al., 1999; Ohman, 2005; Ohman & Mineka, 2001), greater reactivity to this area in typically aggressive individuals may increase the likelihood of these individuals responding aggressively, especially if the threat is perceived to be surmountable. Sober individuals high in self-reported aggression have also been show to misidentify anger in facial cues (Hall, 2006). The effect of alcohol consumption on the processing of emotional expression may therefore be influenced by individual differences in trait levels of aggression.

1.5. Thesis Objectives

1.5.1. Methodology

Both observational (non-experimental) and experimental methods have been used to examine the influence of alcohol consumption on several social cognition outcomes. More specifically, the experimental work in this thesis addresses the influence of acute consumption on emotional face processing accuracy (chapter 2), hostile attribution bias towards facial expressions (chapter 4), and hostile attribution bias towards dyadic social interactions (chapter 5). The cross-sectional observational work addresses whether chronic consumption influence emotional face processing (chapter 3). Triangulating results using these different approaches will improve the strength of evidence in response to the proposed research questions (Heale & Forbes, 2013). When results produce effects or associations in the same direction using similar approaches, bias is reduced, and the reliability of results is subsequently improved (Lawlor et al., 2016).

1.5.2. Statistical Inferences

Statistical inferences throughout this thesis of work were made in accordance with the Tobacco and Alcohol Research Group (TARG) guidelines. Specifically, p values are included as an index of strength of evidence against the null hypothesis (H_0) (Dahiru, 2008) and are interpreted as follows: $p > .10$ indicates no clear evidence against the H_0; $.05 < p < .10$ indicates weak evidence against the H_0; $.001 < p < .05$ indicates moderate evidence; $p < .001$ indicates strong evidence against H_0. These parameters were taken from the suggestions outlined by Sterne and Davey Smith (2001). The studies in this thesis of work were designed with a minimum of 80% statistical power consistent with conventional guidelines (Perugini et al., 2018). This was to ensure that each study was suitably powered to detect an anticipated effect should there be one to detect.

1.5.3. Aims and Hypotheses

This thesis aims to address several research objectives. Chapter 2 will focus on exploring the effect of acute alcohol consumption on emotional face recognition in high and low trait aggressive social drinkers. The primary aim of this chapter is to investigate whether a moderate dose of alcohol impairs the ability of social drinkers to accurately identify the emotion displayed in a face. It is anticipated that consumption will result in a global deficit in emotional face processing consistent with literature that consistently report a global effect of alcohol (Tucker & Vuchinich, 1983). This chapter will also explore whether alcohol influence the processing of specific emotions. Six basic emotional facial expressions: anger, sadness, happiness, disgust, fear, and surprise, will be used. It is anticipated that consumption will influence the recognition of angry and sad facial expressions specifically. These effects were anticipated to be more pronounced in high compared to low trait aggressive drinkers. The influence chronic alcohol consumption has on emotion face processing will be investigated in chapter 3. This chapter will specifically address whether high weekly alcohol consumption predicts poorer global and emotion specific processing deficits amongst regular social drinkers. It is anticipated that non-dependent social drinkers will show a similar profile of emotion processing impairment displayed by an alcohol-dependent individuals in previous research (Donadon & Osorio, 2017). Chapter 4 aims to build on the findings of chapter 2 by exploring how individuals interpret emotional facial expressions following acute alcohol consumption. This chapter primarily addresses whether social drinkers perceive greater hostility in emotional facial expressions when intoxicated. Again, the six basic emotions (anger, sadness, happiness, disgust, fear, surprise) will be used. It is anticipated that there will be a greater hostile attribution bias towards these expressions following alcohol mimicking similar biases reported in research using a forensic

sample (Smeijers et al., 2017). Hostile biases will also be explored at an emotion specific level following alcohol and placebo drinks. Chapter 5 will specifically focus on dyadic social interactions. This chapter aims to build upon the wealth of evidence exploring isolated emotional facial expression by addressing alcohol induced deficits in processing the intentions of others when two people are interacting.

CHAPTER 2: EFFECTS OF ACUTE ALCOHOL CONSMUPTION ON EMOTION RECOGNITION IN HIGH AND LOW TRAIT AGGRESSIVE DRINKERS

Keywords: Acute Alcohol Consumption; Emotional Facial Expressions; Trait Aggression; Emotion Perception Bias & Sensitivity.

The study that forms this chapter was published in the Journal of Psychopharmacology on the 29th June 2020 (DOI: 10.1177/0269881120922951).

2.1. Chapter Overview

The purpose of this chapter was to investigate the acute effects of alcohol on emotional face recognition in individuals with high and low trait aggression. Research suggests that acute alcohol consumption impairs processing of emotional faces. As emotional processing plays a key role in effective social interaction, these impairments may be one mechanism by which alcohol changes social behaviour. Regular non-dependent drinkers, either high or low in trait aggression participated in a double-blind placebo-controlled experiment (N=88, 50% high trait aggressive). Participants attended two sessions. In one they consumed an alcoholic drink (0.4 g/kg) and in the other they consumed a matched placebo. They then completed two computer-based tasks: a six-alternative forced choice task (6AFC) measured global and emotion-specific recognition performance across six primary emotions (anger, happiness, sadness, disgust, fear, surprise), and two-alternative choice tasks (2AFC) measured processing bias of two ambiguously expressive faces (happy-angry/happy-sad). There was evidence of poorer global emotion recognition after alcohol. In addition, there was evidence of poorer sensitivity to sadness and fear after alcohol and also evidence for a reduced response bias towards happiness following alcohol. There was no evidence of any of these effects being more pronounced in high compared to low trait aggressive individuals. These findings suggest that alcohol impairs global emotion recognition. They also highlight a reduced ability to detect sadness and fearful facial expressions. As sadness and fear are cues of submission and distress (i.e., function to curtail aggression), failure to successfully detect these emotions when intoxicated may increase the likelihood of aggressive responding. This coupled with a reduced bias towards seeing happiness may collectively contribute to aggressive behaviour.

2.2. Introduction

Emotional facial expressions are important social cues and non-verbal forms of communication that are considered a fundamental component of effective social interactions (Moriya et al., 2013). In sober individuals, sad and fearful facial expressions are distress cues that promote prosocial behaviour in others and inhibit aggression (Eisenberg et al., 1989; Marsh et al., 2007), whilst angry expressions may reduce socially unacceptable behaviour in some individual. It is therefore plausible that acute alcohol induced deficits in emotion processing will lead to aggressive behaviour. Recent research indicates that acute alcohol consumption can alter the processing of emotional facial expressions. Some evidence exploring the prosocial effects of alcohol on emotion processing report a reduction in the time taken to recognise happy faces following acute consumption (Dolder et al., 2017). Similar research also suggests that happy faces were better recognised following alcohol (Kano et al., 2003). However, it has also been suggested that deficits in emotion processing may be a mechanism involved in increased aggressive behaviour following acute alcohol consumption (Attwood & Munafo, 2014). Some evidence has found that acute alcohol consumption impairs the overall ability to process emotional facial expressions, irrespective of the emotion displayed (i.e., global emotion processing) (Tucker & Vuchinich, 1983). At an emotion specific level, an increased bias towards perceiving angry faces (in ambiguous negative facial morphs) has been reported following acute alcohol consumption (Attwood, Ataya, et al., 2009). This altered processing is likely to have a meaningful impact on behaviour, as a bias towards seeing anger may increase perceived provocation, which is a primary driver of aggression (Giancola et al., 2002). In addition, research has demonstrated a decreased sensitivity towards perceiving sadness following acute alcohol consumption (Craig et al., 2009). This has further implications for alcohol-related aggression, as sadness is an indicator of

submission (Hart, 2011), which may curtail aggression. More recent data from our group has found weak evidence supporting an anger bias after alcohol consumption, but effect sizes are small (Khouja et al., 2019).

The majority of this research has been conducted using unselected samples (i.e., social drinkers). It is important to consider individual differences amongst alcohol consumers as only a small proportion of alcohol consumers reliably display alcohol-related aggression (Attwood & Munafo, 2014). In a review of emotion recognition in forensic samples, research suggest that violent offenders generally demonstrate emotion recognition deficits (Chapman et al., 2018). The most consistently reported deficits are a reduced accuracy for disgusted faces (Robinson et al., 2012; Seidel et al., 2013) and a reduced sensitivity towards fearful faces (Gillespie et al., 2015; Schonenberg et al., 2014; Schonenberg et al., 2013). The observation of the impairment to the processing of fearful faces is particularly relevant to aggressive behaviour as these are considered to be cues of submission likely to curtail aggressive responding (Marsh et al., 2007). There is also mixed evidence of an anger perception bias (a potential driver of aggression) amongst this population with some suggesting that violent offenders have a bias (Schonenberg & Jusyte, 2014) and others suggesting no bias (Hoaken et al., 2007). Taken together, this evidence suggests that individuals with heightened levels of trait aggression tend to process emotional facial expression poorly and may have specific deficits in processing specific emotional expressions. In the wider population, it is well established that higher levels of trait aggression are predictive of alcohol-related aggression after provocation (Bailey & Taylor, 1991; Eckhardt & Crane, 2008; Giancola, 2002a; Giancola et al., 2005; Giancola et al., 2002; Giancola & Zeichner, 1995; Miller et al., 2009; Moeller et al., 1998; Tremblay et al., 2008). Furthermore, sober individuals high in self-reported aggression are more likely to misidentify anger in facial cues (Hall, 2006). Therefore, it is

reasonable to speculate that alcohol may exacerbate these effects in high trait aggressive individuals, which in turn may contribute to the higher levels of alcohol-related aggression in these groups.

2.3. Aims

This study investigated the effects of alcohol consumption on emotional face processing in social alcohol drinkers who were either high or low in trait aggression. Emotion recognition of six emotions (anger, happiness, sadness, disgust, fear, surprise) were measured using a six-alternative forced choice (6AFC) task. In addition, two-alternative forced choice (2AFC) tasks presenting angry-happy and happy-sad emotional morphs were used to test bias in the interpretation of ambiguous emotional expressions. It was hypothesised that there would be a global deficit in emotion processing, an increased sensitivity towards perceiving anger, and a decreased sensitivity towards perceiving sadness in the 6AFC task following alcohol compared to placebo. It was also hypothesised that there would be an increased bias towards angry emotions and a reduced bias towards sad emotions in the 2AFC tasks following alcohol compared to placebo. These effects were anticipated to be more pronounced in high compared to low trait aggressive drinkers.

2.4. Methods

2.4.1. Participants

Social drinkers (N = 88, 50% male) were recruited from the University of Bristol (staff and students) as well as the general population by means of existing email lists, poster advertisement and word of mouth. Participants were either high or low in trait aggression, defined by a score on the Anger Expression Index subscale (AXi) of the State-Trait Anger Expression Inventory–2 (STAXI-2) (*see 2.4.4 Materials).* Equal numbers of participants were recruited per trait group. The inclusion criteria included: good physical and psychiatric health (self-report), aged between 18-40 and speak English as first language or equivalent level of fluency. To avoid including participants with little/no drinking experience or undiagnosed alcohol dependence, only individuals that consumed between 5 and 35 alcoholic UK units per week if female or between 10 and 50 alcoholic UK units per week if male were included. One UK unit equals one 25 ml single measure of spirit (ABV 40%), or a third of a pint of beer (ABV 5-6%) or half a standard (175 ml) glass of red wine (ABV 12%) (NHS, 2018). The exclusion criteria were any individuals that reported a strong familial history of alcoholism defined as one or more immediate relatives (e.g., parents and/or siblings) or more than one other relatives (e.g., cousin, grandparents), that reported a history of psychiatric disorder (including drug addiction). Exclusions also include any individual that reported consuming alcohol 24 hours prior to testing or if their breath alcohol concentration (BrAC) was above zero (tested on arrival, *see 2.4.5 Procedures*), and if they weighed less than 50kg if female or 60kg if male. Participants gave signed informed consent prior to taking part in the study. On completion, participants were reimbursed £20 or course credits (where appropriate). The study was approved by the University of Bristol's Faculty of Science Human Research Ethics

Committee (reference: 26011747361). The study protocol was pre-registered on the Open Science Framework (doi: 10.17605/OSF.IO/YV392).

2.4.2. Design

A double-blind placebo-controlled experimental design was used. This comprised one within-subject factor of drink (alcohol, placebo) and one between-subject factor of trait aggression (high, low; 50% male in each group). For the 6AFC measures, an additional within-subject factor of emotion was included (anger, happiness, sadness, disgust, fear, surprise). Participants completed the alcohol and placebo conditions on separate days (at least one week apart). Session order was counterbalanced with equal numbers of participants in each order group. Participants were allocated session orders in advance of the study using random number generator software (www.randomizer.org).

2.4.3. Drink

Drinks were prepared by a research collaborator who was independent of data collection and therefore drink delivery was double-blind. Alcohol content was dependent on participant weight. An upper limit of 90 kg was set so that participants weighing more than 90 kg received the same drink as a 90 kg participant. The alcoholic drinks were mixed using one-part vodka (37.5% ABV) to three parts tonic water. The dose used was 0.4 grams of alcohol per kg of body weight (g/kg (Attwood, Ataya, et al., 2009; Craig et al., 2009). Placebo drinks were matched volume tonic water. In order to mask the taste of alcohol, drinks were chilled and flavoured with lime cordial (40 ml) prior to serving. The inside rim of the glass was sprayed twice with a vodka mist.

2.4.4. Materials

2.4.4.1. Computerised Tasks.

The images used in both tasks are composite (i.e., prototypical) images created from photographs of 12 young male adults expressing each of 6 emotions (angry, sad, happy, disgust, fear, surprise). The photographs were taken in a booth painted Munsel N5 grey which was illuminated with 3 Verivide F20 T12/D65 daylight simulation bulbs in high-frequency fixtures (Verivide, UK), which reduced the effects of flicker. Using established techniques (Tiddeman et al., 2001), the 12 images for each emotional expression were delineated with 172 feature points, which allowed colour and shape information to be averaged across faces to produce a full prototypical exemplar expression for each emotion (see Figure 2.1a). Trials in both tasks begins with a centrally-displayed fixation cross. A 350 × 457 pixel face stimulus is then presented for 150 ms, followed by a noise mask for 250 ms in order to prevent after-image effects. Tasks were run using E-Prime 2.0 Pro software, on a standard computer with QWERTY keyboard.

2.4.4.1.1. Six-alternative forced choice task (6AFC).

Six 15-image morph sequences were created, one for each emotion (anger, happiness, sadness, disgust, fear, surprise). An overall emotionally ambiguous face was generated by averaging the exemplars for each emotional expression. A linear continuum of 15 images were produced for each emotion ranging from an emotionally ambiguous prototype to the full emotional intensity (see Figure 2.1b). An emotionally ambiguous prototype was used instead of neutral, as experimental evidence suggests this gives a better approximation of the centre of emotional face-space (Skinner & Benton, 2010). Theses stimuli have been used in a series of published research (e.g., Attwood et al., 2017; Bamford et al., 2015; Griffiths et al., 2015). On each trial, a single image from the 90 available was presented for 150 ms (backward masked), and participants were required to identify

the emotion as quickly and as accurately as possible, by using the mouse to click on the most appropriate descriptor from an array of descriptors displayed on-screen (angry, sad, happy, disgust, fear, surprise). The descriptor array appeared on-screen for 10,000 ms, or until the participant responded. Each image was presented twice, giving 180 trials in total. The measures of interest were proportion of total hits (i.e., global emotion processing accuracy), emotion specific hit rates (i.e., emotion specific processing accuracy) and false alarms (i.e., misattribution of a particular emotion for another).

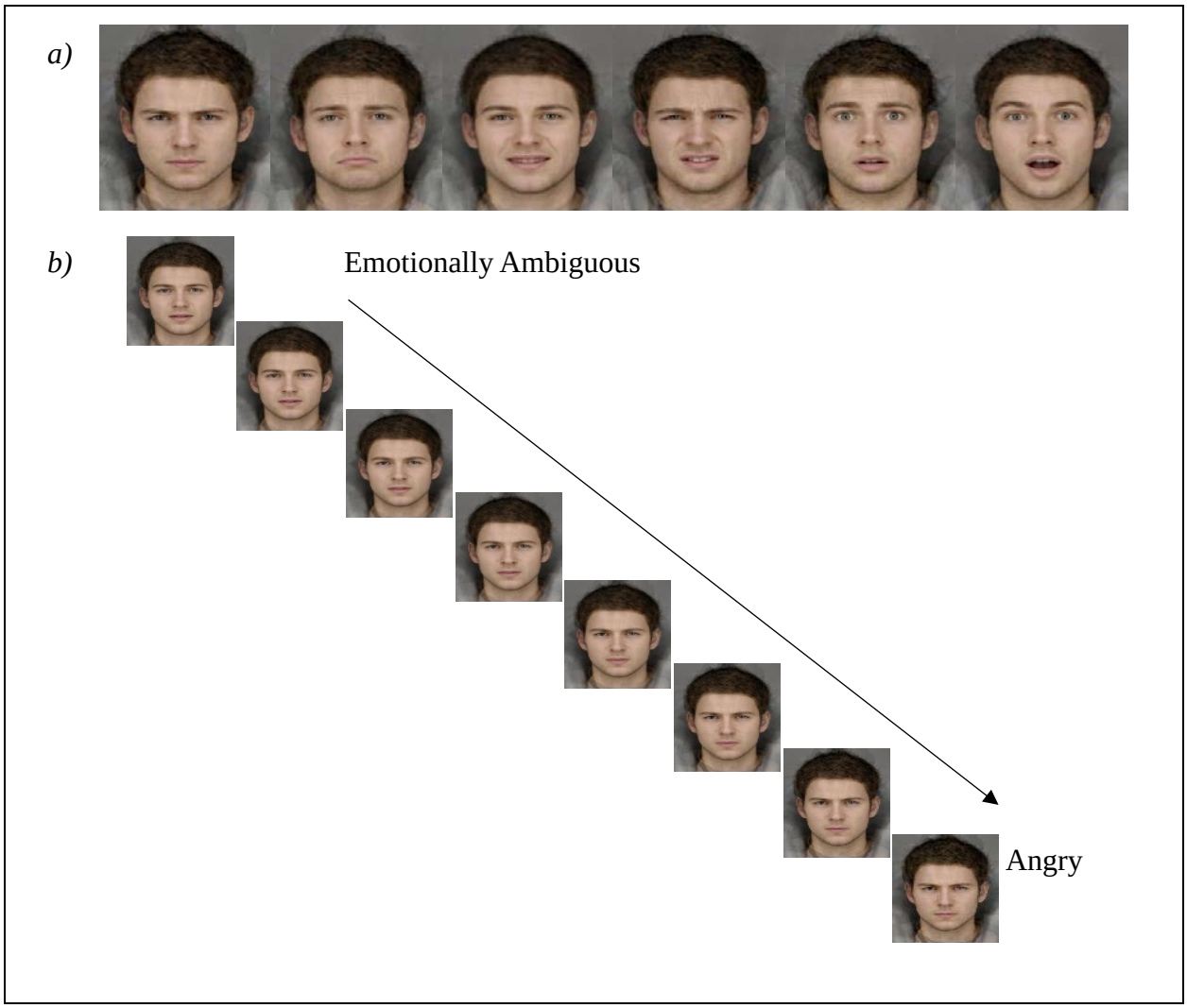

Figure 2.1: a) Full intensity examples of the 6 basic emotions used in the 6AFC task. Facial expressions are angry, sad, happy, disgust, fear, surprise from left to right. b) 15-image morph sequence for the angry emotion. Stimuli range from emotionally ambiguous to full emotion intensity.

2.4.4.1.2. Two-alternative forced choice task (2AFC).

Two 2AFC tasks were used including a happy-angry and a happy-sad continuum. For each of these tasks, a 15-image morph sequence was created, which runs from one full emotional exemplar to another (e.g., unambiguously happy to unambiguously angry / unambiguously happy to unambiguously sad) (see Figure 2.2). The full exemplar images (i.e., 100% emotion intensity) are used as endpoints to create a linear morph sequence of images that change incrementally from happy to angry in one task version and happy to sad in the other. On each trial, a frame from this morph continuum was presented for 150 ms (backward masked), and participants were required to identify whether the emotion was happiness or anger (task1) or happiness or sadness (task2), by pressing designated keys on the keyboard. Each image is presented three times, giving 45 trials in total for each 2AFC task. The primary outcome was an estimate of the point on the 15-image continuum at which the participant was equally likely to respond happy or angry/happy or sad (the *balance point*). The balance point for each emotion continuum was estimated by calculating the number of happy responses proportionate to the number of trials; greater values indicate a bias towards happy emotions (lower values indicate a bias towards angry/sad emotions).

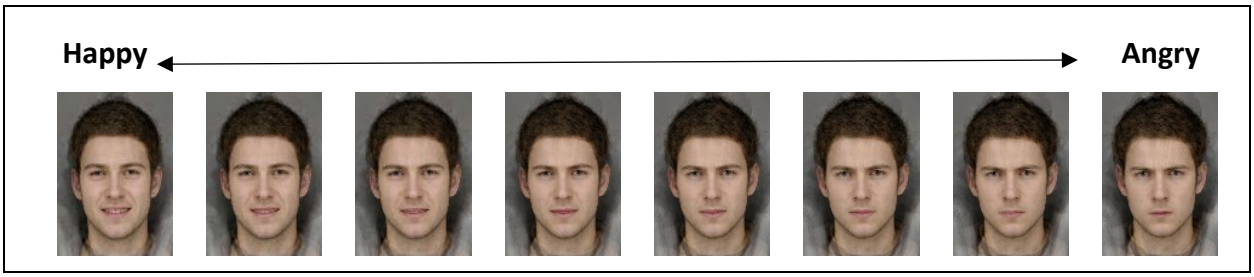

Figure 2.2: 15-image morph sequence used in the Happy-Angry 2AFC task. The images range from the full intensity example of the happy emotion along a linear continuum to the full intensity example of the angry emotion.

2.4.4.2. Questionnaire Measures.

Trait aggression was measured using the anger expression index subscale (AXi) of the State-Trait Anger Expression Inventory (STAXI-2) (Spielberger, 1999). (Forgays et al., 1997). Normative data for the STAXI-2 scale are based on samples of normal adults (n=1,644) ranging from 16-63 years old; these data show a mean score of 32.9 (SD = 13.4) for the AXi subscale. High and low trait aggression groups were defined by a score above the 60th percentile and below the 40th percentile on this subscale, respectively. Other questionnaire measures included the State Anger Subscale (S-Ang) of the STAXI-2 (Spielberger, 1999), Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988), Biphasic Alcohol Effects Scale (BAES) (Martin et al., 1993) and Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993).

2.4.5. Procedures

Prior to testing, participants completed the STAXI-2 online. Individuals that met the inclusion criteria (i.e., high or low in trait aggression) were invited to take part in the study via email. Participants were required to attend two sessions, at least one week apart. In one they received an alcoholic drink and in the other they received a matched placebo (order counterbalanced). On arrival at the first session, participants were given the opportunity to read the information sheet again and ask questions, before providing written informed consent. Participants then completed a short screening procedure to verify eligibility. Weight was also recorded during screening. Participants were breathalysed (Draeger AlcoDigital 3000 Breathalyzer) to confirm zero BrAC before each testing session. Weight information was passed to the collaborator to prepare the drink. Participants then completed the baseline questionnaires (AUDIT, PANAS, BAES and S-Ang). Participants were given 10 minutes to consume all of their drink and a further 10 minutes to sit quietly to allow for absorption. Following this, participants

were instructed to complete the 6AFC and the two 2AFC tasks (fixed order). They then complete questionnaires again (i.e., PANAS, BAES, S-Ang) and provided another BrAC reading. Before leaving, participants signed a safety card. They were offered the opportunity to stay behind until they felt any effects of alcohol had worn off and were offered a taxi home. At the end of session two, participants were debriefed and reimbursed.

2.4.6. Sample Size Calculation

The sample size was based on previous findings using a between-subjects design (Craig et al., 2009), which indicated an effect size of $d = 1.0$ for the difference between alcohol and placebo on sadness recognition (M = 0.14, SD = 0.02; M = 0.12, SD = 0.02, respectively). This indicated that a total sample size of 46 participants would be required to achieve 90% power at an alpha level of 5%. As the present study included a between-subjects factor, we planned to recruit sufficient numbers in each group to achieve this level of power to observe a main effect of alcohol. However, this was likely to be an inflated effect size, so a more conservative effect size estimate of $d = 0.7$ was used. Based on this estimate, 88 participants were required in each drink condition in a between-subjects design to achieve 90% power at an alpha level of 5%. As the alcohol/placebo condition in the present study was within-subjects, we considered this to be a conservative estimate. Therefore, 44 participants were recruited per trait group (total n = 88). This would provide 90% power to detect an effect size of $dz = 0.5$ (alcohol vs. placebo) within each trait group.

2.4.7. Statistical Analysis

Statistical analyses were conducted using IMB SPSS Statistics (version 24). Total hits (i.e., 6AFC data) and balance points (i.e., 2AFC data) were assessed for outliers using boxplots. Participant data were removed if scores were 1.5 times greater than the interquartile range (*Ns reported in the results)*. Normality was assessed using skewness and kurtosis z-score statistics.

There were no violations of normality unless otherwise stated. Homogeneity of variance was assessed using Levene's test of equality and no violations (e.g., $p < .05$) were detected unless otherwise stated. Mauchly's Test of Sphericity was used and where $p < .05$, Greenhouse-Geisser corrected statistics are reported.

For 6AFC data, a task programming error meant that the presentation of the surprise emotion was compromised. This error meant that two full intensity surprise images and 28 emotionally ambiguous images (i.e., 5% along the continuum between 'emotional ambiguity' to 'full intensity' surprise) were presented to the participants when completing the task. As a result, the responses to the full intensity images were excluded from emotion specific analyses and the surprise emotion was recategorised as emotionally ambiguous. For the analysis of total hit rate, all erroneous surprise responses were completely removed.

The total hits data were analysed using a 2 drink (alcohol, placebo) × 2 aggression (high, low) mixed model ANOVA. It was pre-registered that anger and sadness specific hits and false alarms, would be analysed separately using 2 drink (alcohol, placebo) × 2 aggression (high, low) mixed ANOVAs. It was later decided that using a signal detection theory (SDT) approach to calculate measures of response sensitivity and bias from emotion specific hit and false alarm data would be more appropriate. According to SDT, response sensitivity reflects the ability to discriminate between the presence of a specific emotion from noise (i.e., the absence of the target emotion), whereas response bias measures the preference for a specific emotion (Macmillan & Creelman, 2005). This allows us to investigate whether there is a genuine deficit in processing a specific emotion (i.e., sensitivity) or whether there is a tendency to see an emotion regardless of whether it is there (i.e., bias). Therefore, a measure of response sensitivity and bias was calculated for both angry and sad emotions using the 6AFC proportion hit rate (p(H)) and false alarm (p(FA))

data. The non-parametric *A'* (Macmillan & Creelman, 2005; Pollack & Norman, 1964) was used as a measure of sensitivity and was calculated using the formula outlined in Stanislaw and Todorov (1999). This was preferred to the parametric *d'* measure of sensitivity as the signal (i.e., presence of the target emotion) and noise (i.e., absence of target emotion) distributions were not normal (Swets, 1986). The *A'* scores typically range from 0 (i.e., emotions cannot be recognised from noise) to 1 (i.e., emotions are distinguishable from noise). The non-parametric *B″* (Grier, 1971) was used as a measure of response bias. With scores ranging from -1 (i.e., a response bias in favour of *emotion present*) to +1 (i.e., a response bias in favour of *emotion not-present*); a score of zero indicates no response bias. Response sensitivity and bias scores were analysed using 2 drink (alcohol, placebo) x 2 trait aggression (high, low) mixed ANOVAs. In addition to the primary focus on anger and sadness processing, sensitivity and bias scores for the remaining four emotions were explored using the same statistical model. The 2AFC data were analysed using 2 drink (alcohol, placebo) × 2 aggression (high, low) mixed model ANOVAs.

State anger (i.e., S-Ang) questionnaire data was analysed using a 2 drink (alcohol, placebo) × 2 time (pre-consumption, post-consumption) ANOVA. Mood (i.e., PANAS) and biphasic alcohol effects (I.e., BAES) questionnaire data were analysed using 2 drink (alcohol, placebo) × 2 aggression (high, low) × 2 time (pre-consumption, post-consumption) mixed model ANOVAs. Interactions were explored in post-hoc analyses using t-tests.

2.5. Results

The data that form the basis of the results are available from the data.bris Research Data Repository (http://data.bris.ac.uk/data/), DOI: 10.5523/bris.33syxpzss1thw20b8daw2safr9).

2.5.1. Participant Characteristics

A total of 88 (50% male) were recruited and tested. Data from one participant were removed from all analyses due to randomisation error. Participants included in the analyses (*n*=87; 49.4% male) were between the ages of 18-39 (*M* = 23.0, *SD* = 4.6) and weighed between 51-106kg (*M* = 70.0, *SD* = 12.3). AUDIT scores ranged from 3 to 25 (*M* = 10.6, *SD* = 5.2). When asked on completion of the study, 28.7% of participants believed they had consumed alcohol when the drink was a placebo. In comparison, 95.4% believed they had consumed alcohol when the drink contained alcohol.

2.5.2. Emotional Facial Expression Processing (6AFC)

2.5.2.1. Total Hits.

Two outliers were removed from the total hits analysis (n = 85; male = 48.2%; high trait aggression = 51.8%). Inclusion of these outliers resulted in no substantial differences in findings. There was strong evidence for a main effect of drink (F [1, 83] = 10.42, p =.002, η_p^2 = .112) with fewer hits following alcohol compared to placebo. There was no clear evidence of a main effect of trait aggression (F [1, 83] = .45, p = .506, η_p^2 = .005) or a drink by trait aggression interaction (F [1, 83] = 1.41, p = .239, η_p^2 = .017) (see Figure 2.3).

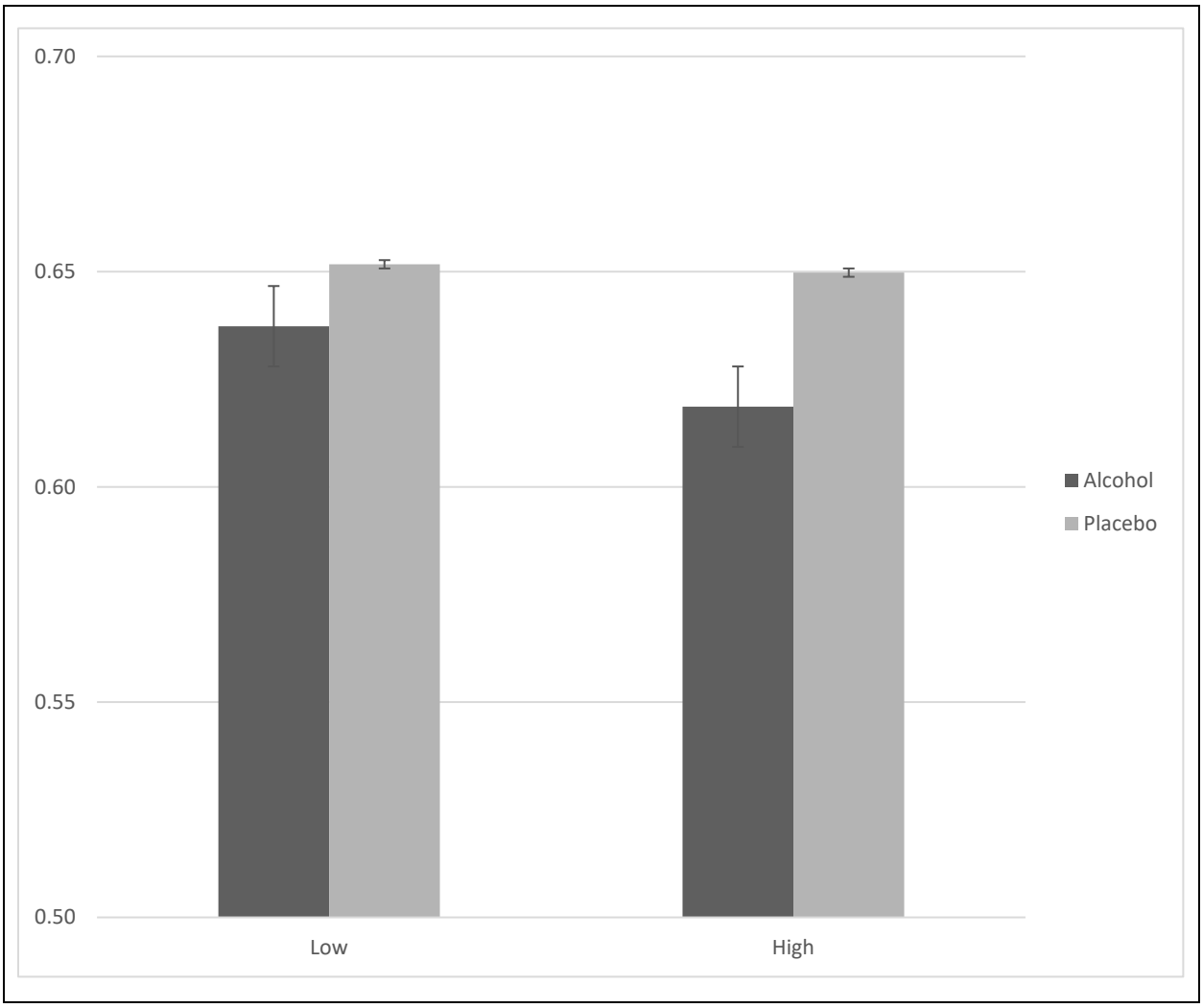

Figure 2.3: Total hit rate scores (6AFC) in high compared to low trait aggressive individuals following both alcoholic and placebo drinks. Error bars represent standard error.

2.5.2.2. Response Sensitivity.

Descriptive statistics for sensitivity scores can be found in Table 2.1. There was modest evidence of a main effect of drink for sadness (*F* [1, 83] = 6.51, p = .013, η_p^2 = .073) and fear (*F* [1, 83] = 4.62, p = .034, η_p^2 = .053). These results demonstrate a reduced sensitivity towards sadness and fear following alcohol compared to placebo. There was weak evidence of a main effect of drink for disgust (*F* [1, 83] = 3.25, p = .075, η_p^2 = .038) also showing a reduced sensitivity following alcohol compared to placebo. There was no evidence of a main effect of drink for anger

or happy emotions (*ps* > .371). There was modest evidence of a main effect of trait aggression for sadness (F [1, 83] = 6.26, p = .014, η_p^2 = .070) and disgust (F [1, 83] = 5.41, p = .022, η_p^2 = .061). These results show a reduced sensitivity towards sad and disgust faces in high compared to low trait aggressive individuals. There was weak evidence of a main effect of trait aggression for anger (F [1, 83] = 3.63, p = .060, η_p^2 = .042) showing that high compared to low trait aggressive individuals demonstrate a reduced sensitivity. There was no clear evidence of a main effect of trait aggression for happy or fear (*ps* > .398), or for an interaction effect for angry, sad, happy, disgust, and fear (*ps* > .172).

2.5.2.3. Response Bias.

Descriptive statistics for bias scores can be found in Table 2.1. There was evidence of a main effect of drink for happiness (F [1, 83] = 5.92, p = .017, η_p^2 = .067) showing a reduced bias towards happiness following alcohol compared to placebo. There was no clear evidence of a drink main effect for anger, sad, disgust, and fear (*ps* > .302). There was modest evidence of a main effect of trait aggression for disgust (F [1, 83] = 4.97, p = .028, η_p^2 = .057) showing an increased bias towards disgust in high compared to low trait aggressive individuals. There was no clear evidence of a main effect of trait aggression for anger, sad, happy, and fear (*ps* > .268), or of a drink x trait aggression interaction for all emotions (*ps* > .391).

Table 2.1: Scores are mean A' (sensitivity) and B″ (bias) for each emotion (anger, sadness, happiness, disgust, fear) in high and low trait aggressive individuals; standard error in parentheses.

			Alcohol	Placebo
Sensitivity	**Anger**	**High**	.903 (.005)	.903 (.006)
		Low	.913 (.005)	.916 (.004)
	Sad	**High**	.907 (.006)	.916 (.003)
		Low	.919 (.004)	.928 (.003)
	Happy	**High**	.891 (.005)	.902 (.005)
		Low	.888 (.007)	.891 (.017)
	Disgust	**High**	.906 (.009)	.913 (.009)
		Low	.927 (.005)	.936 (.005)
	Fear	**High**	.580 (.040)	.631 (.041)
		Low	.552 (.043)	.564 (.04)
Bias	**Anger**	**High**	.825 (.027)	.819 (.028)
		Low	.842 (.025)	.840 (.025)
	Sad	**High**	.388 (.076)	.441 (.063)
		Low	.396 (.070)	.434 (.085)
	Happy	**High**	.501 (.075)	.333 (.078)
		Low	.348 (.079)	.259 (.093)
	Disgust	**High**	.353 (.077)	.319 (.077)
		Low	.513 (.055)	.547 (.052)
	Fear	**High**	.634 (.044)	.670 (.038)
		Low	.615 (.049)	.599 (.046)

NOTE: A' (Macmillan & Creelman, 2005; Pollack & Norman, 1964) is a measure of response sensitivity and B" (Grier, 1971) is a measure of response bias. The A' scores ranged from 0 (i.e., emotions cannot be recognised from noise) to 1 (i.e., emotions are perfectly distinguishable from noise). B" scores ranging from -1 (i.e., a response bias in favour of always seeing the emotion as present) to +1 (i.e., a response bias in favour of not seeing the emotion as present).

2.5.3. Emotional Facial Expression Balance Point (2AFC)

2.5.3.1. Happy - Angry.

Four outliers were removed (n=83; 49.4% male; 53.0% high trait aggressive). Inclusion of these outlier resulted in no substantial differences in findings. Descriptive data for happy-angry balance points can be seen in Figure 2.4. There was no clear evidence for a main effect of drink (F [1, 81] = .15, p = .702, η_p^2 = .002) or trait aggression (F [1, 81] = .49, p = .486, η_p^2 = .006), or for a drink by trait aggression interaction (F [1, 81] = .99, p = .322, η_p^2 = .012) on happy-angry balance points.

2.5.3.2. Happy – Sad.

Three outliers were removed (n=84; 47.6% male; 51.2% high trait aggressive). Inclusion of these outlier resulted in no substantial differences in findings. Descriptive data for happy-sad balance point scores can be seen in Figure 2.4. There was weak evidence for a main effect of drink (F [1, 82] = 3.49, p = .065, η_p^2 = .041) indicating a bias towards sad faces following alcohol (M = 6.33, SE = .17) compared to placebo (M = 6.61. SE = .15). There was also weak evidence for a main effect of trait aggression (F [1, 82] = 2.86, p = .095, η_p^2 = .034) indicating a bias towards sad faces in high (M = 6.23, SE = .20) compared to low high trait aggressive individuals (M = 6.71, SE = .20). There was no clear evidence for a drink x trait aggression interaction (F [1, 82] = .81, p = .371, η_p^2 = .010) on happy-sad balance points.

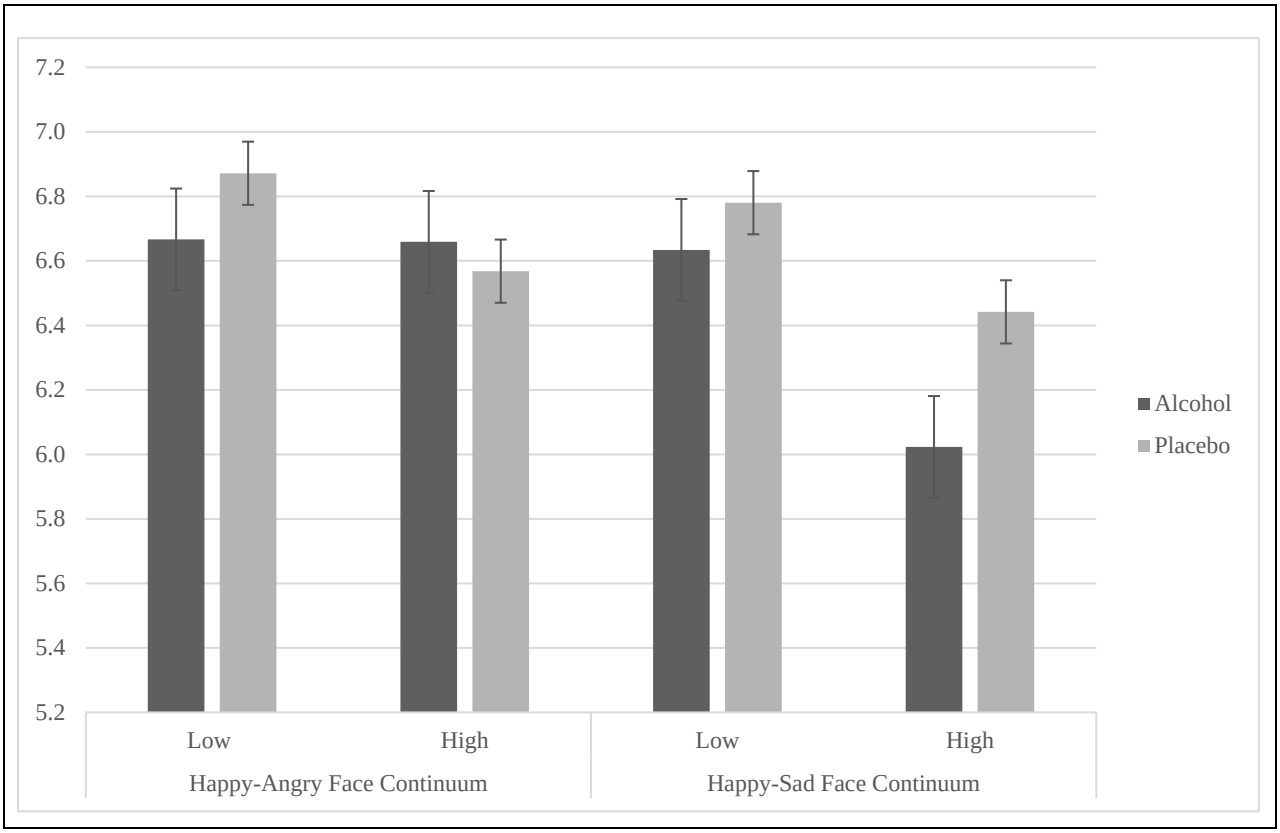

Figure 2.4: Emotion balance-points following alcohol and placebo drinks in high and low trait aggressive drinkers. A greater score indicates a bias for happy faces, whilst lower scores indicates a bias for angry/sad faces. Error bars are standard error.

2.5.4. Sensitivity Analyses

Six participants weighed more than 90kg and so received a dose of alcohol less than .4g/kg (as 90kg was used as a maximum cut off). Sensitivity analyses excluding these participants were conducted for comparison. Total hits, response sensitivity and response bias (i.e., 6AFC) results did not substantially differ. Similarly, Happy-Angry and Happy-Sad 2AFC results did not substantially differ (results not shown).

2.5.5. Questionnaire Measures

Descriptive data for all questionnaire measures (i.e., S-Ang, PANAS, BAES) can be found in Table 2.2. There was no clear evidence for a main effect of drink or time, or for a drink x time interaction for S-Ang (*ps* > .266).

There was no clear evidence for main effects of drink or trait aggression (*ps* > .582*)* on positive affect (i.e., PANAS). The was strong evidence for a main effect of time (F [1, 85] = 10.04, p = .002, η_p^2 = .106) with lower positive affect post consumption. There was no clear evidence for any interactions (two-way and three-way) between drink, time and trait aggression on positive affect (ps > .178*),* or for a main effect of drink (p = .633) on negative affect. There was weak evidence for a main effect of time (F [1, 85] = 3.13, p = .080, η_p^2 = .036) with lower negative affect post consumption. There was also strong evidence for a main effect of trait aggression (F [1, 85] = 11.94, p = .001, η_p^2 = .123) with greater negative affect reported by high trait aggressive individuals. There was no clear evidence for any interactions (two-way and three-way) between drink, time and trait aggression on negative affect (ps > .132*).*

There was no clear evidence for a main effect of drink or trait aggression (*ps* > .343*)* on self-report levels of alcohol induced stimulation (i.e., BAES). There was modest evidence for a main effect of time (F [1, 85] =6.17, p = .015, η_p^2 = .068) with greater levels of self-reported

stimulation pre consumption. There was no clear evidence for any interactions (two-way and three-way) between drink, time and trait aggression on self-reported levels of stimulation (p*s* > .198*).* For self-reported levels of sedation, there was strong evidence for a main effect of time (*F* [1, 85] = 43.71, *p* < .001, η_p^2 = .340) with greater levels of self-reported sedation post consumption, and a main effect of trait aggression (*F* [1, 85] = 9.04, *p* = .003, η_p^2 = .096) with greater levels reported by high trait aggressive individuals. There was also weak evidence for a main effect of drink (*F* [1, 85] = 3.38, p = .069, η_p^2 = .038) with reduced levels reported following alcohol. There was strong evidence for an interaction between drink and time (*F* [1, 85] = 10.55, *p* = .002, η_p^2 = .110). To explore this interaction further post hoc t-tests were conducted. These analyses suggest that self-report levels of sedation increase post drink-consumption (compared to pre-consumption) following both alcohol *(t* [86] =6.47, *p* < .001) and placebo *(t* [86] =3.02, *p* = .003). There was no clear evidence for any interactions (two-way and three-way) between drink, time and trait aggression on self-reported levels of sedation (*ps* > .181).

Table 2.2: Scores are means for all questionnaire measures (i.e., S-Ang, PANAS, BAES); standard error in parentheses.

				Pre-Consumption	Post-Consumption
S-Ang			**Alcohol**	16.1 (.3)	16.2 (.3)
			Placebo	16.1 (.3)	15.9 (.3)
PANAS	**Positive Affect**	**Low**	**Alcohol**	27.3 (1.1)	24.6 (1.1)
			Placebo	26.8 (1.0)	23.6 (1.2)
		High	**Alcohol**	25.7 (1.1)	23.5 (1.1)
			Placebo	26.5 (1.0)	24.3 (1.2)
	Negative Affect	**Low**	**Alcohol**	11.0 (.5)	11.1 (.4)
			Placebo	11.2 (.5)	10.9 (.4)
		High	**Alcohol**	13.1 (.5)	12.8 (.4)
			Placebo	13.3 (.5)	12.0 (.4)
BAES	**Stimulant**	**Low**	**Alcohol**	29.3 (2.0)	23.8 (1.8)
			Placebo	26.0 (2.0)	22.6 (2.3)
		High	**Alcohol**	27.9 (1.9)	25.9 (1.8)
			Placebo	29.4 (2.0)	25.7 (2.2)
	Sedative	**Low**	**Alcohol**	9.3 (1.8)	17.0 (2.3)
			Placebo	10.7 (1.5)	14.2 (2.1)
		High	**Alcohol**	14.3 (1.8)	26.9 (2.3)
			Placebo	14.4 (1.5)	18.8 (2.0)

NOTE: State Anger Subscale (S-Ang) of the State-Trait Anger Expression Inventory (STAXI-2) *(Spielberger, 1999), Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988), Biphasic Alcohol Effects Scale (BAES) (Martin et al., 1993). S-Ang higher scores indicate greater state levels of aggressions; higher PANAS scores reflect greater positive and negative affect; higher BAES scores indicate greater self-reported levels of sedation and stimulation.*

2.6. Discussion

This study investigated whether emotion processing of facial expressions was affected by acute alcohol consumption in high and low trait aggressive individuals. Results show fewer total hits (i.e., 6AFC) following alcohol compared to placebo highlighting a global deficit in emotion processing following alcohol compared to placebo. This is consistent with Tucker and Vuchinich (1983) who also found poorer global emotion recognition following acute alcohol consumption. As the recognition of emotional faces is a key factor involved in successful social interactions (Moriya et al., 2013), this reduced ability to accurately identify emotional expressions may contribute to misinterpretation of emotional states and intentions of others, leading to poorer social function when intoxicated (Adolphs & Tusche, 2017). Comparisons can be made with forensic population studies similarly highlighting poorer global emotion processing in offenders compared to non-offenders (Chapman et al., 2018), suggesting that acute alcohol consumption may mimic the impairment seen in individuals that comment violent offences. However, the effect of acute alcohol consumption on global emotion processing reported in this study was not found to be more pronounced in high compared to low trait aggressive individuals. At an emotion specific level, SDT measures indicated a reduced sensitivity towards sad and fear expressions following alcohol consumption. There was also weak evidence suggesting reduced sensitivity to disgusted emotional expressions. These findings have social relevance, as fear and sadness in particular are considered to be signals of distress and submission (Blair, 2005; Hart, 2011) which can curtail aggression (e.g., signals avoidance and low confrontation to potential aggressors). Therefore, a decrease in sensitivity to these emotions following the consumption of alcohol, increases the likelihood of aggressive behaviour. This is consistent with past literature that similarly report a decreased sensitivity towards sadness following alcohol (Craig et al., 2009). Again, comparisons can be made

with forensic samples that show reduced accuracy for disgusted faces (Robinson et al., 2012; Seidel et al., 2013) and a reduced sensitivity towards fearful faces (Gillespie et al., 2015; Schonenberg et al., 2014; Schonenberg et al., 2013). This again suggests that alcohol consumption may mimic the emotion specific impairment shown by violent offenders to signals of distress (i.e., fearful faces) and may explain why intoxicated individuals respond aggressively. There was no evidence to suggest that these effects of alcohol on emotion sensitivity differed in high and low trait aggressive individuals. However, results did show that high trait aggressive individuals demonstrate a reduced sensitivity towards sad and disgust faces, further supporting the idea that typically aggressive individuals miss socially relevant distress cues. Response bias (i.e., *B"*) is an indicator of preference for one emotion over the other remaining emotions (Grier, 1971). Results show a reduced bias towards happy emotions following alcohol compared to placebo. As happiness is considered to be a positive emotion and is often the most easily identifiable emotion (Calvo & Beltran, 2013) a reduction in happiness response bias following alcohol may function to promote aggressive behaviour.

There was no evidence of alcohol-related bias towards angry faces in the happy-angry 2AFC task. This is consistent with Khouja et al. (2019) who similarly report no anger bias in happy-angry facial morphs but contradicts (Attwood, Ataya, et al., 2009) who do report an anger bias in negative facial morphs (i.e. anger-disgust facial morphs). A possible explanation for these differences surrounds the face-morph continuum used. Positive emotions (i.e., happiness) are reported to be more easily identified than negative emotions (i.e., anger and disgust) (Calvo & Beltran, 2013). It is therefore possible that negative face morphs (i.e., angry-disgust) result in an anger bias but the inclusion of a positive emotion (i.e., happy-angry) do not. Further investigation using alternative morphed pairs of emotional stimuli will allow for this discrepancy to be better

understood. Similarly, there was no evidence of a change in bias in happy-angry facial morphs in high compared to low trait aggressive individuals. There was however weak evidence to suggest alcohol led to a sadness perception preference in the happy-sad facial morph. However, it is unclear whether this captures a reduced happiness or increased sadness perceptual bias. Further exploration of bias using alternative 2AFC emotion facial morphs (i.e., sad-anger) will help disentangle this in future research. Similarly, high trait aggressive individuals showed a preference for sad over happy faces in the happy-sad facial morph. Again, it is difficult to conclude whether this reflects a bias towards sadness or a reduction in bias towards happiness.

This study used a double-blind placebo-controlled experimental design. The placebo manipulation had a relatively low success rate with only a third of participants believing they had consumed alcohol in the placebo condition. As a result, there was a limiting lack of control over the anticipated effects of alcohol. Evidence has shown that the expectation of alcohol leads to individuals adapting their behaviour to compensate for the anticipated effects of alcohol (Marczinski & Fillmore, 2005). As the majority of participants receiving a placebo drink in this study were not convinced the drink contained alcohol, these compensatory mechanisms due to expectancy were arguably reduced. This compared to the alcohol condition where participants were expecting alcohol and receiving it, may have led to a dampened effect of alcohol due to the compensatory mechanisms associated with expectancy. However, evidence surrounding the placebo effect in alcohol research is mixed largely due to the variation in drinking experiences that shape each individuals' expectancies (Testa et al., 2006). To address these limitations, future emotion processing research could explore the specific pharmacological effects of alcohol using a balanced placebo design (Sayette et al., 1994). This design would allow an anti-placebo (i.e., alcohol administered but not expected) vs. control (i.e., no alcohol administered and not expected)

comparison which best models a pure pharmacological effect. It would also allow effects that are due to expectancy to be tested (i.e., placebo vs. control).

Another line of inquiry for future research surrounds chronic alcohol consumption and emotional processing. Research has shown that excessive chronic consumption produces pronounced emotion recognition impairment in alcohol dependent samples (Kornreich et al., 2001; Maurage, Campanella, Philippot, Martin, et al., 2008). Specifically, this research highlights that alcohol dependent individuals are poorer at processing and recognising emotional facial expressions. It is likely that non-dependent chronic drinkers display similar impairment (albeit to a lesser extent). Chapter 3 specifically tests this by exploring the associations between weekly units of alcohol consumed and emotion processing ability (sustained over a minimum period of 5 years). It is important to establish whether increased alcohol consumption over time (i.e., chronic consumption) in non-dependent drinkers is linked to emotion processing deficits considering these are key forms of non-verbal communication (Moriya et al., 2013) and are capable of influencing behaviour (Eisenberg et al., 1989; Klinnert, 1983; Marsh et al., 2007).

2.6.1. Conclusion

Findings suggest that acute alcohol consumption disrupts the processing of emotional facial expressions. These have several implications as emotional expressions are important social cues that function to guide behaviour. Failure to accurately process these cues may lead to maladaptive behaviour. At an emotion specific level, alcohol decreases the ability to detect distress and submissive social cues, such as sad and fearful emotional expressions. This may contribute to alcohol related aggression as these emotional expressions tend to signal avoidance to the perceiver which in turn curtail aggression. Therefore, failure to detect these cues when intoxicated are likely to contribute to aggressive responding.

CHAPTER 3: ASSOCIATIONS BETWEEN CHRONIC ALCOHOL CONSUMPTION AND EMOTIONAL FACIAL EXPRESSION PROCESSING

Keywords: Chronic Alcohol Consumption, Emotional Face Processing, Emotion Perception Bias & Sensitivity

3.1. Chapter Overview

The purpose of this chapter was to investigate whether chronic alcohol consumption (i.e., units of alcohol consumed per week for a minimum period of 5 years) was associated with emotional face processing deficits in non-dependent drinkers. Research shows that alcohol dependent individuals are poorer at accurately identifying emotions, require greater emotion intensity and take longer to make accurate judgements, compared to healthy controls. As the ability to process emotions accurately plays a key role in effective social interaction, impairments due to chronic consumption may be one mechanism by which social behaviour is changed. Regular non-dependent drinkers (n=188) that report having consumed alcohol for a sustained period of at least 5 years participated in a cross-sectional online study. The number of typical drinks consumed per week were self-reported and this information was used to estimate the number of weekly alcoholic units typically consumed. Information surrounding binge drinking behaviour (i.e., never/occasionally, monthly or weekly) was also recorded to explore the influence this has on emotional face processing. Following this, participants completed an emotion recognition task measuring performance across six primary emotions (anger, happiness, sadness, disgust, fear, surprise). Outcome measures were global processing accuracy and emotion specific response sensitivity and bias. There was no evidence of an association between chronic alcohol consumption and global emotion processing accuracy. Longitudinal birth cohort studies have reported similar findings when assessing the influence of binge drinking in late adolescence/early adulthood (16-23 years old) on later global emotion processing accuracy (age 24). At an emotion specific level, there was evidence for a reduced sensitivity towards sadness as units of alcohol consumed per week increased. This association is important considering this emotion has been found to curtail aggression (i.e., it cues distress and submission). Findings are somewhat limited by the cross-

sectional design used. Future research should assess associations between chronic alcohol consumption and emotional face processing longitudinally.

3.2. Introduction

Chapter 2 highlighted that acute alcohol consumption influenced both global and emotion specific emotion processing amongst social drinkers. Key findings include a reduced sensitivity towards sadness and fear, and a reduced bias towards happiness. Similar acute alcohol research also reports a decreased sensitivity towards sadness following alcohol (Craig et al., 2009). As previously mentioned, recent reviews indicate that both chronic (Donadon & Osorio Fde, 2014) and acute alcohol consumption (Attwood & Munafo, 2014) can alter the processing of emotional facial expressions. Beck and Heinz (2013) report that a substantial proportion of alcohol dependent men display violent behaviour (between 16-50% depending on the severity of violence and the age of the individual). Research using general population samples has also suggested that long term/habitual alcohol consumption is associated with an increased likelihood of committing an act of aggression (Bye, 2007; Wells et al., 2000). Whilst research has indicated an association between chronic alcohol use and aggression, it is clear that this behaviour is not an inevitable consequence as it does not occur in all alcohol dependent individuals or all chronic alcohol consumers (Beck & Heinz, 2013). It is possible that aggression is a result of the long-term effects of alcohol consumption on cognitive, biological and social mechanisms associated with aggression. It is likely that alcohol-related changes in emotional processing will influence behaviour. Deficits in emotional face processing have been documented in alcohol abusers (Foisy et al., 2007; Townshend & Duka, 2003). Specifically, Donadon and Osorio (2017) demonstrate that individuals that are alcohol dependent are poorer at accurately identifying emotions, require greater emotion intensity and take longer to make accurate judgements, compared to healthy controls. What

remains unclear is whether chronic alcohol consumption within the general population (i.e., non-dependent samples) is associated with poorer emotion recognition. Research has also demonstrated a tendency for alcoholics to erroneously identify emotions such as sadness as anger when processing facial expressions suggesting an anger perception bias (Frigerio et al., 2002; Philippot et al., 1999). As this research demonstrates a potential disruption to the processing of sad faces, it is therefore likely that sadness perception specifically will be influenced by chronic consumption as similar deficits when seeing this emotion have also been reported in acute alcohol studies (Attwood, Ohlson, et al., 2009; Craig et al., 2009).

3.3. Aims

This study aims to test whether chronic alcohol consumption is associated with poorer emotional face recognition in a non-dependent sample. Participants recruited were weekly alcohol consumers over a sustained period for a minimum of 5 years (in order to capture frequent consumption over time). This was selected so that individuals had at least 5 years exposure to alcohol. Units of alcohol consumed per week were measured to assess chronic alcohol consumption. In addition, frequency of binge drinking was recorded to explore whether this was associated with emotion processing impairment. It was also anticipated that greater weekly alcohol consumption and frequent binge drinking over a minimum period of 5 years would disrupt the processing of specific emotions, consistent with prior research amongst alcoholic drinkers. As the effects of alcohol use on cognition differ depending on gender (Ganguli et al., 2005; Stampfer et al., 2005) and age (Neafsey & Collins, 2011), analyses were adjusted for these. Age was particularly important given that chronic consumption is likely to be dependent on age increasing (i.e., weekly alcohol consumption over a longer period of time due to increased exposure). Analyses were also adjusted for trait levels of aggression as evidence suggests an association

between habitual drinking and aggression (Bye, 2007; Wells et al., 2000). Outcome measures included emotion recognition of six emotions (happy, sad, angry, disgust, surprise and fear) measured using a six-alternative forced choice (6AFC) task.

3.4. Methods

3.4.1. Participants

Weekly alcohol consumers were recruited and screened for eligibility via Prolific (https://www.prolific.ac/). Participants were eligible if they were weekly alcohol consumers reporting sustained consumption for a minimum of 5 years (self-report). Other eligibility criteria included good physical and psychiatric health (self-report), aged 18 and over and speak English as first language or equivalent level of fluency. Participants were not eligible if they reported a strong familial history of alcoholism defined as one or more immediate relative (parent, sibling) or more than one other relative (e.g., cousin, grandparent), and if they reported a history of psychiatric disorder (including drug addiction). Participants read the study information and gave online consent. On completion, participants were reimbursed £2. The study was approved by the University of Bristol's Faculty of Science Human Research Ethics Committee (reference: 20012092022). The study protocol was pre-registered on the Open Science Framework (doi: 10.17605/OSF.IO/2TFWJ)

3.4.2. Design

A cross-sectional observational design was used. The primary independent variable was weekly units of alcohol consumed (i.e., chronic alcohol consumption). This was estimated by gathering information on how many *typical* alcoholic drinks each participant consumed during a week (see 3.4.3 *Measures and Materials)*. Frequency of binge drinking (defined as how frequent 6 or more alcoholics units if female, or 8 or more if male, were consumed on a single drinking

occasion in the last year) was used an exploratory independent variable. The primary outcome measure was global emotional processing accuracy (i.e., total hits 6AFC). Additional outcome variables of response sensitivity and response bias for each of the 6 basic emotions (i.e., happy, sad, angry, disgust, fear and surprise) were used to explore emotion specific associations with chronic alcohol consumption and frequency of binge drinking. Age, gender, education and trait aggression information were controlled for. Analyses were adjusted to account for these and compared with the unadjusted models (to determine the degree of influence).

3.4.3. Measures and Materials

3.4.3.1. Six-alternative forced choice task (6AFC)

The images used in the 6AFC are composite (i.e., prototypical) images created from photographs of 12 young male adults taken under controlled conditions. Six 15-image morph sequences have been created, one for each emotion (happy, sad, angry, disgust, surprise and fear). These run along a linear continuum from an emotionally ambiguous prototype to the full emotional intensity (90 face stimuli in total). Each trial in both tasks begins with a centrally displayed fixation cross. A face stimulus is then presented at random for 150 ms, followed by a noise mask for 250 ms in order to prevent after-image effects. In this emotion recognition task, participants are required to identify the emotion represented in the face as quickly and as accurately as possible, by using the mouse to click on the most appropriate descriptor from an array of descriptors displayed on-screen (happy, sad, angry, disgust, surprise and fear). The descriptor array appears on-screen for 10,000 ms, or until the participant responds. Each image is presented twice, giving 180 trials in total. This task takes approximately 10-12 minutes to complete.

3.4.3.2. Questionnaires

To measure chronic alcohol consumption, participants were asked to record how many standard alcohol drinks they consumed during a typical week. These drinks include Bottle (5%/330ml), Can (5.5%/500ml), Low Pint (3.6%/568ml), High Pint (5.2%/568ml), Small wine (12%/125ml), Standard wine (12%/175ml), Large wine (12%/250ml), Shot (40% - 25ml), Alcopop (5.5%/275ml). Units per week were then calculated using the following formula: strength (ABV) x volume (ml)/1,000 (NHS, 2018). Frequency of binge drinking was measured using the following question: *How often have you had 6 or more units if female, or 8 or more if male, on a single occasion in the last year? ([0] - never, [1] - less than monthly, [2] - monthly, [3] - weekly, [4] – daily or almost daily).* Binge drinking responses were used to derive a three-level ordinal variable with categories including Never/Occasional (comprising Never and less than monthly), Monthly, and Weekly (comprising weekly and daily or almost daily) binge drinkers consistent with the categorisation described by (Mahedy et al., 2020). Trait aggression was measured using the anger expression index subscale (AXi) of the State-Trait Anger Expression Inventory (STAXI-2) (Spielberger, 1999). Sociodemographic factors including age, gender, education (Degree or equivalent Higher education, A-Level or equivalent, GCSE grades A*-C or equivalent, other qualification, don't know/no qualification) were measured. Finally, the Alcohol Use Disorders Identification Test (AUDIT) (Babor, Higgins-Biddle, Saunders, & Monteiro, 2001) was used to measure hazardous and harmful drinking.

3.4.4. Procedure

Each participant completed a single online session lasting approximately 20 minutes. The study was accessed through Prolific (https://www.prolific.ac/) and hosted by Gorilla™ (hosted by Microsoft Azure; https://gorilla.sc/). Prolific handled screening and participants were asked to

verify eligibility. Eligible participants were directed to Gorilla and were given time to read a study information page and a consent statement before providing informed consent. Following this they completed a series of short questions measuring weekly units of alcohol consumption, and demographic information including gender, age and education. The STAXI-2 and AUDIT questionnaires were also completed. Participants then completed the 6AFC task. Finally, participants were required to read debriefing information and asked to provide final consent before being reimbursed.

3.4.5. Sample Size Determination

The sample size was determined from a meta-analysis exploring facial emotion recognition and alcohol use disorder (AUD) (Bora & Zorlu, 2017). Facial recognition was impaired in AUD compared to healthy controls (d = 0.65, 95% confidence interval (CI) = 0.42–0.89). As the present study uses a correlational design, this effect size was transformed to f^2 = .11 in accordance with Cohen (1988). Sample size was calculated using this effect size estimate and indicated that 121 participants would be required to achieve 95% power at alpha level 5%. As this effect size was obtained in an alcohol dependent sample and the present study aims to investigate chronic alcohol consumption within the general population (i.e., non dependent sample), the effect size was reduced by a third (f^2 = .07) to ensure a more conservative sample size was calculated. This indicated that 188 participants would be required to achieve 95% power at alpha level 5%.

3.4.6. Statistical Analysis

Data from 199 participants was screened during data collection. Participant data was removed if it was incomplete (n=7). Total hit rates were also screened for outliers. Four participants were removed from analysis as z-scores indicated total hits scores +/- 3 standard deviations from the mean. Further inspection of these outlier data indicates that the 6AFC was completed in less

than 8 minutes. Data from 188 participants were analysed. A regression model was used to assess associations between chronic alcohol consumption (i.e., weekly units of alcohol consumed) and global emotion processing deficits (i.e., 6AFC total hits). Multivariate normality, homoscedasticity and multicollinearity assumptions were satisfied unless otherwise stated. Models were also produced to test associations between chronic alcohol consumption and emotion specific response sensitivity and response bias for each of the 6 basic emotions (i.e., happy, sad, angry, disgust, fear and surprise). A signal detection theory (SDT) approach to calculate measures of response sensitivity and bias from emotion specific hit and false alarm data was used. According to SDT, response sensitivity reflects the ability to discriminate between the presence of a specific emotion from noise (i.e., the absence of the target emotion), whereas response bias measures the preference for a specific emotion (Macmillan & Creelman, 2005). This allows the investigation in to whether there is a genuine deficit in processing a specific emotion (i.e., sensitivity) or whether there is a tendency to see an emotion regardless of whether it is there (i.e., bias). Therefore, a measure of response sensitivity and bias was calculated for both angry and sad emotions using the 6AFC proportion hit rate (p(H)) and false alarm (p(FA)) data. The non-parametric *A'* (Macmillan & Creelman, 2005; Pollack & Norman, 1964) was used as a measure of sensitivity and was calculated using the formula outlined in Stanislaw and Todorov (1999). This was preferred to the parametric *d'* measure of sensitivity as the signal (i.e., presence of the target emotion) and noise (i.e., absence of target emotion) distributions were not normal (Swets, 1986). The *A'* scores typically range from 0 (i.e., emotions cannot be recognised from noise) to 1 (i.e., emotions are distinguishable from noise). The non-parametric *B″* (Grier, 1971) was used as a measure of response bias. With scores ranging from -1 (i.e., a response bias in favour of seeing the target emotion) to +1 (i.e., a response bias in favour of not seeing the target emotion); a score of zero indicates no response bias. In

addition, these analyses were adjusted for age, gender, education and trait aggression. To explore the role frequent binge drinking (i.e., Never/occasional, monthly, weekly) plays in emotional face processing, regression models were used to test associations between binge drinking and global (i.e., total hits), as well as emotion specific (i.e., response sensitivity and bias). Never/occasional binge drinking was compared to monthly and weekly binge drinking in each model. Age, gender, education and trait anger were controlled for in these models.

3.5. Results

3.5.1. Participant Characteristics

Regular drinkers (n =188; 60.6% male) were recruited and tested. They were between the ages of 18-69 (*M* = 32.54, *SD* = 11.0). AUDIT scores ranged from 1 to 30 (*M* = 8.6, *SD* = 5.2). 69.7% of participants were educated to degree (or equivalent) level or higher, 16.5% had A-Level (or equivalent) qualifications, 9.6% had grade A*-C GCSE's (or equivalent) qualifications, 3.2% reported other qualifications, and 1.1% reported no qualification or that they didn't know. Trait anger scores ranged from 10 to 40 (*M* =18.7, *SD* = 5.8*).* 51.6% of participants reported never/or occasionally binge drinking, 31.9% reported binge drinking monthly and 16.5% reported binge drinking weekly.

3.5.2. Global Emotion Processing Accuracy

The model estimates of the association between chronic alcohol consumption and binge drinking with global emotion processing accuracy (i.e., total hits) are displayed in Table 3.1. There was no evidence for an association between units of alcohol consumed per week or binge drinking with global emotion processing (*p >.516*). Figure 3.1 displays the association between units of alcohol consumed per week and total hits, and Figure 3.2 displays the association for each type of binge drinker (i.e., between never/occasional, monthly and weekly binge drinkers) and total hits. The model adjusted for the influence of age, gender, education and trait anger. There was strong evidence for an association between gender and global emotion processing (*ps <.008*). Estimates suggest that males are 4.2% poorer at accurately recognising emotions displayed in a face compared to females. There was no evidence of an association between age, education and trait anger (*ps >.226*).

Table 3.1: Regression model estimates, 95 % Confidence intervals, and p-values for global emotional face recognition accuracy (i.e., total hits).

Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	.738	.579 – .897	**<0.001**	.733	.573 – .893	**<0.001**
Age	-.001	-.002 – .001	.275	-.001	-.002 – .001	.226
Gender [Female= 0, Male= 1]	-.042	-.073 – -.011	**.008**	-.042	-.074 – -.011	**.008**
Education [Degree or equivalent Higher education]	-.061	-.208 – .087	.416	-.060	-.208 – .088	.423
Education [A Level or equivalent]	-.037	-.188 – .114	.631	-.037	-.189 – .115	.630
Education [GCSEs grades A*-C or equivalent]	-.022	-.178 – .133	.775	-.025	-.180 – .131	.754
Education [Other qualifications]	-.077	-.245 – .091	.366	-.075	-.243 – .094	.382
Trait Anger	.0001	-.003 – .003	.958	.00005	-.003 – .003	.969
Units of Alcohol per week	-.0004	-.001 – .001	.516			
Binge Drinking [Monthly]				.011	-.023 – .045	.536
Binge Drinking [Weekly]				-.002	-.045 – .041	.921
Observations		188			188	
R^2 / R^2 adjusted		.062 / .021			.063 / .015	

Note: Age in years, Gender [Female= 0, Male= 1], Education [Unknown/No Qualification =0, Degree or equivalent Higher education =1, A Level or equivalent= 2, GCSEs grades A*-C or equivalent =3, Other qualifications =4], *Trait Anger Subscale of the STAXi-2, Units of Alcohol calculated from self-reported consumption data of standard drinks typically consumed per week, Binge Drinking [Never/Occasional = 0, Monthly = 1, Weekly =2]. Total hits are the proportion of correctly identified emotions.*

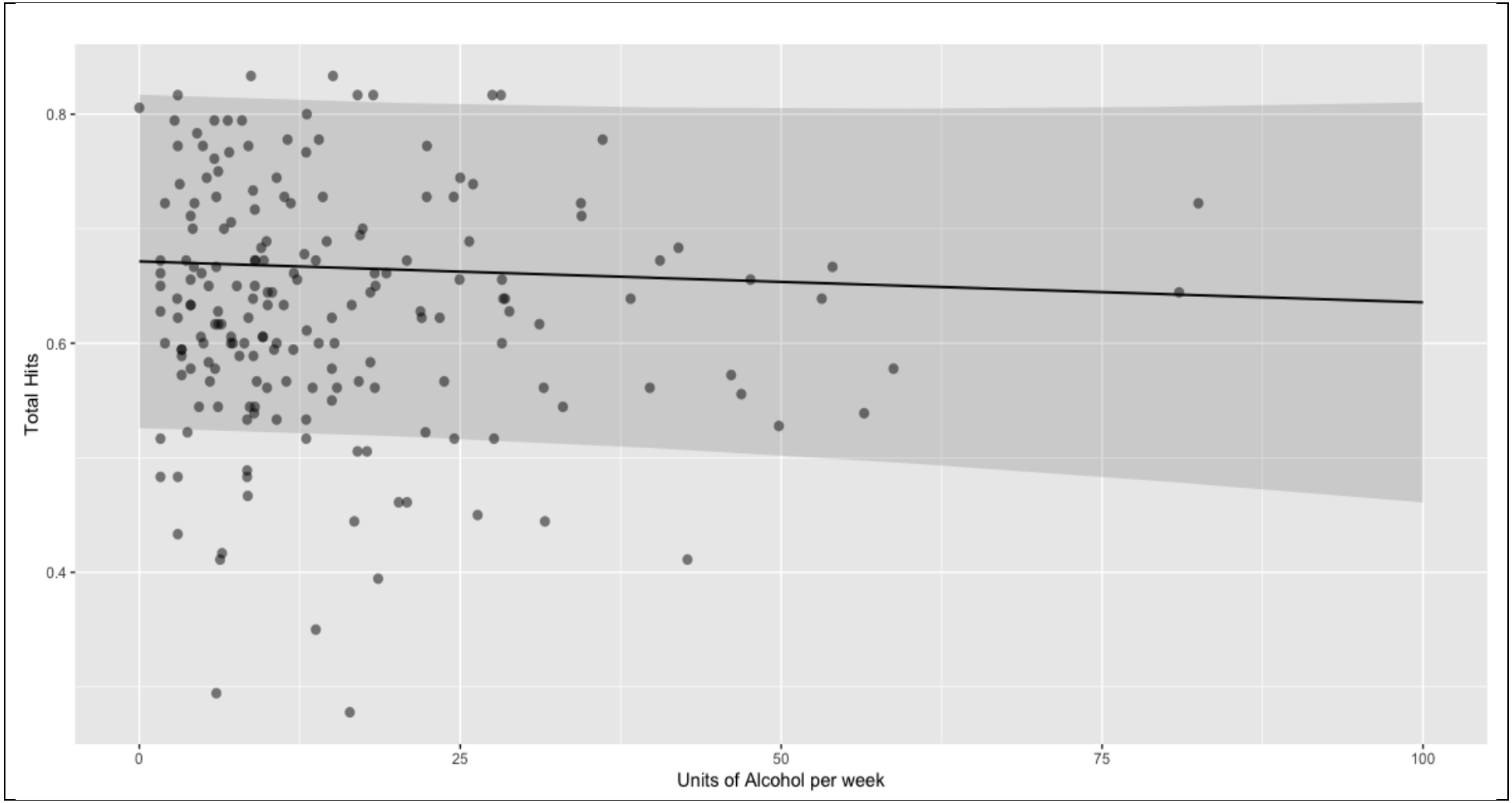

Figure 3.1: The association of units of alcohol per week with global emotion processing accuracy (i.e., total hit rate). Regression lines are estimated from the model.

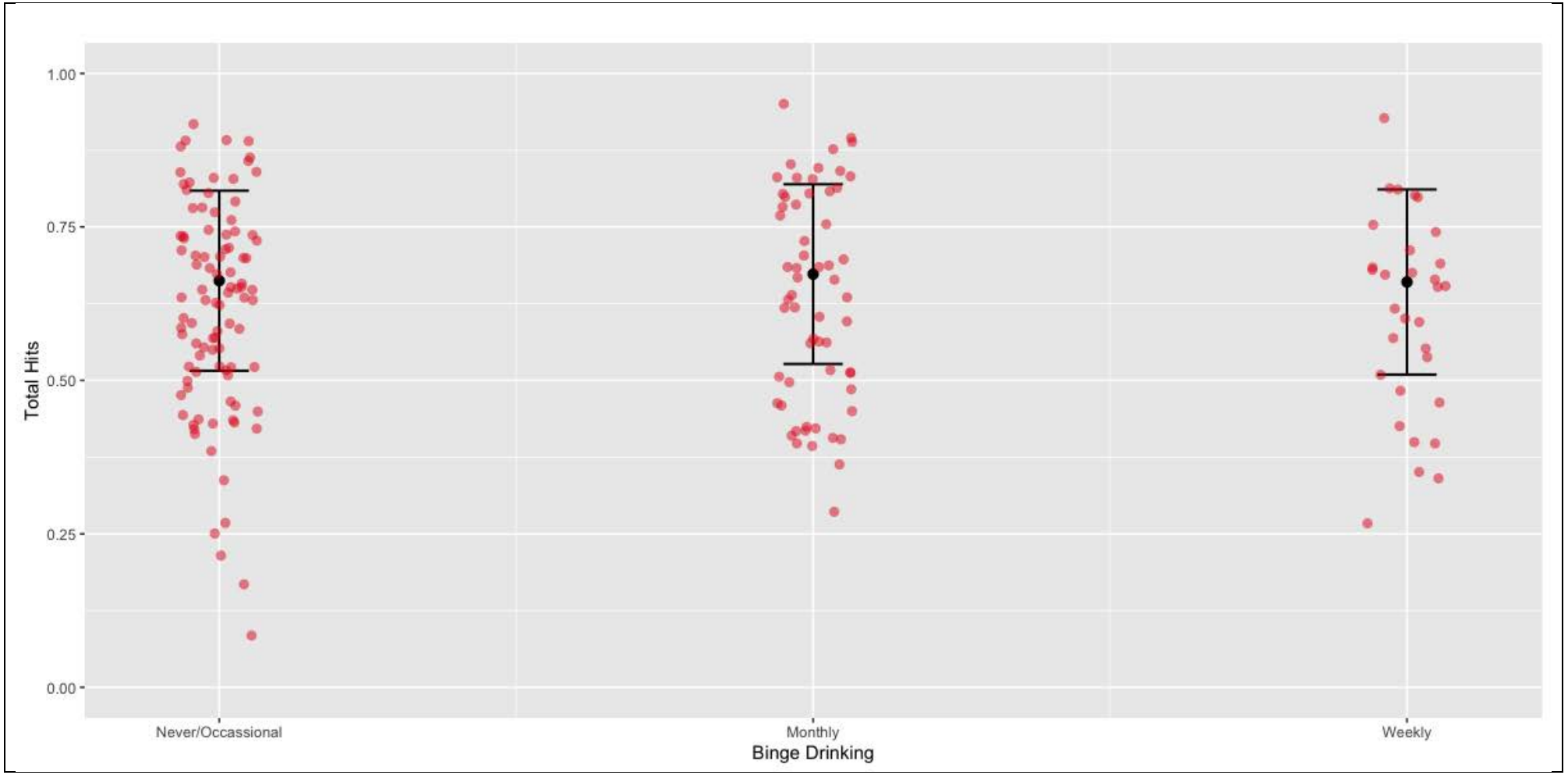

Figure 3.2: The association between binge drinking and global emotion processing accuracy (i.e., total hit rate). Error bars represent 95% confidence intervals.

3.5.3. Emotion Specific Response Sensitivity

The model estimates of the association between chronic alcohol consumption and emotion specific response sensitivity (i.e., A' Prime) for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise) can be found in Table 3.2. There was modest evidence for an association between sadness sensitivity and units of alcohol consumed per week ($p = .039$). Estimates for this model suggest that every unit increase in alcohol consumed per week is associated with a .001 decrease in sadness sensitivity. This observable trend can be seen in Figure 3.3. There was no evidence for an association for the remaining emotions (Anger, Happy, Disgusted, Fear, Surprise); Figure 3.3 displays the association between emotion processing sensitivity and units consumed per week for each emotion. Models were adjusted for the influence of age, gender, education and trait anger. There was modest evidence for an association between gender and emotion specific response sensitivity for disgusted, fearful and surprised faces ($ps < .046$) and weak evidence for this association when seeing happy faces ($p = .059$). Estimates suggest that males are less sensitive to these emotions. There was no evidence of an association for age, education and trait anger for any emotion ($ps > .106$).

Table 3.2: Regression model estimates, 95 % Confidence intervals, and p-values for emotion specific response sensitivity (i.e., A' Prime) for each emotion.

	Anger			**Happy**			**Sad**			**Disgust**			**Fear**			**Surprise**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	.907	.771 – 1.044	**<0.001**	.911	.812 – 1.009	**<0.001**	.933	.849 – 1.017	**<0.001**	.961	.861 – 1.060	**<0.001**	.986	.673 – 1.299	**<0.001**	.906	**.841 – .970**	**<0.001**
Age	.0004	-.001 – .002	.528	.0001	-.001 – .001	.788	.0003	-.0005 – .001	.481	-.001	-.002 – .0002	.106	-.002	-.005 – .001	.163	-.0003	-.001 – .0003	.278
Gender [Female= 0, Male= 1]	-.016	-.043 – .011	.243	-.019	-.038 – .001	**.059**	-.011	-.028 – .005	.174	-.020	-.039 – -.0004	**.046**	-.081	-.142 – -.020	**.010**	-.016	-.028 – -.003	**.014**
Education [Degree or equivalent Higher education]	-.050	-.177 – .077	.435	-.019	-.110 – .072	.683	-.044	-.122 – .034	.271	-.025	-.117 – .067	.593	-.156	-.447 – .134	.290	-.004	-.064 – .056	.904
Education [A Level or equivalent]	-.035	-.166 – .095	.592	.007	-.087 – .101	.882	-.030	-.110 – .050	.464	-.029	-.124 – .066	.546	-.101	-.400 – .197	.503	-.004	-.066 – .058	.899
Education [GCSEs grades A*-C or equivalent]	-.037	-.170 – .097	.587	-.009	-.106 – .087	.846	-.037	-.119 – .045	.378	-.035	-.132 – .062	.476	-.068	-.373 – .238	.663	.014	-.049 – .077	.658
Education [Other qualifications]	-.041	-.185 – .103	.574	-.045	-.149 – .058	.389	-.033	-.122 – .055	.460	-.022	-.127 – .082	.674	-.153	-.483 – .177	.362	-.043	-.111 – .025	.214
Trait Anger	.001	-.002 – .003	.570	-.00003	-.002 – .002	.970	.001	-.001 – .002	.235	-.0001	-.002 – .002	.907	-.002	-.007 – .003	.403	-.0001	-.001 – .001	.787
Units of Alcohol per week	-.0004	-.001 – .001	.406	-.0001	-.001 – .001	.727	-.001	-.001 – -.00003	**.039**	-.0004	-.001 – .0002	.208	.002	-.0004 – .004	.105	.00002	-.0004 – .0005	.946
Observations		188			188			188			188			188			188	
R^2 / R^2 adjusted		.022 / -.021			.052 / .010			.055 / .012			.051 / .009			.079 / .038			.073 / .032	

Notes: Age is years, *Gender [Female= 0, Male= 1], Education [Unknown/No Qualification =0,* Degree or equivalent Higher education =1, A Level or equivalent= 2, GCSEs grades A*-C or equivalent =3, Other qualifications =4], *Trait Anger Subscale of the STAXi-2, Units of Alcohol calculated from self-reported consumption data of standard drinks typically consumed per week. A' Prime (Macmillan & Creelman, 2005; Pollack & Norman, 1964) is a measure of response sensitivity, and scores typically range from 0 (i.e., emotions cannot be recognised from noise) to 1 (i.e., emotions are perfectly distinguishable from noise.*

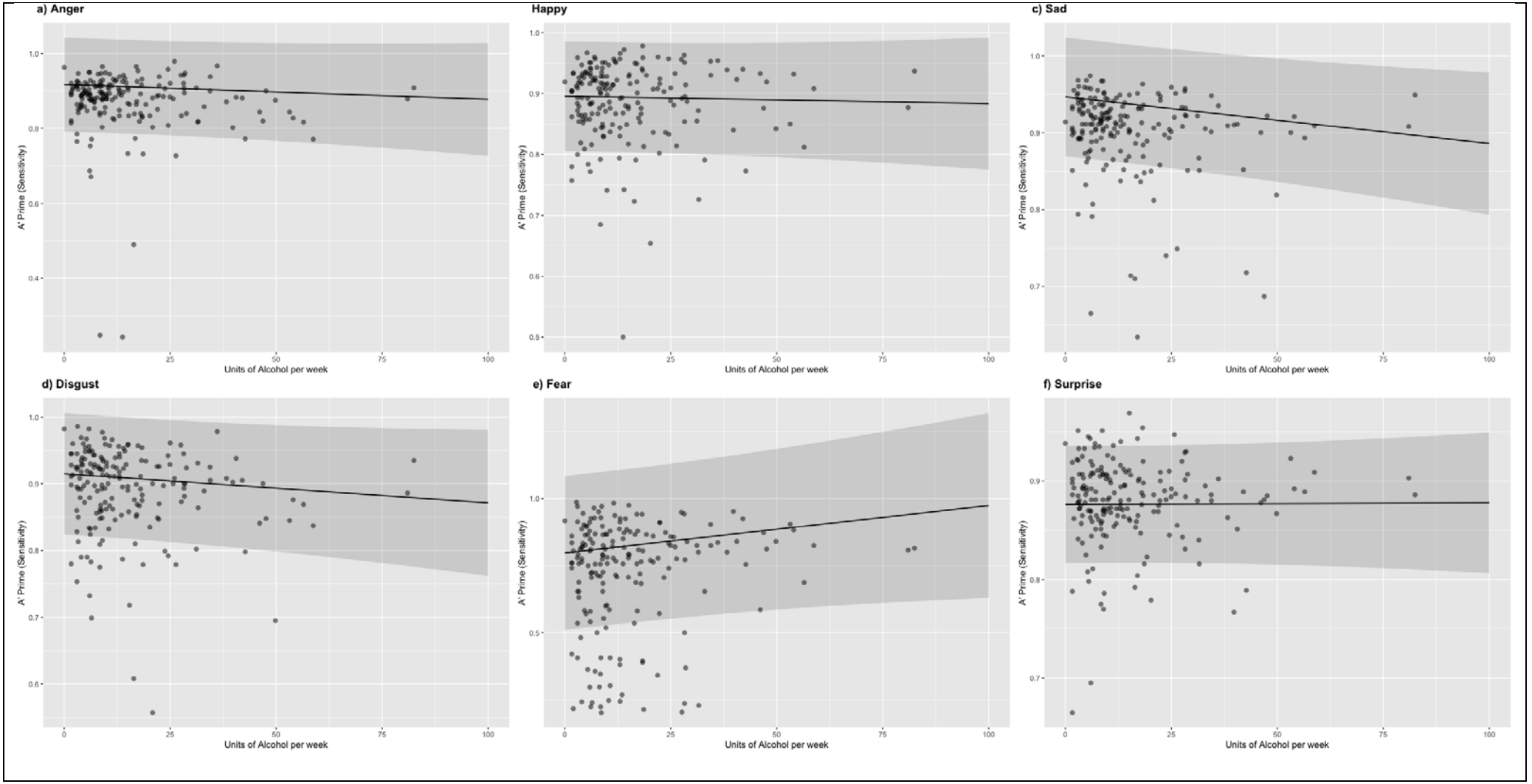

Figure 3.3: The association of units of alcohol per week with emotion specific response sensitivity (i.e., A' Prime) for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise). Regression lines are estimated from each emotion model.

The model estimates of the association between binge drinking and emotion specific response sensitivity (i.e., A' Prime) for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise) can be found in Table 3.3. Figure 3.4 displays observable differences between never/occasional, monthly and weekly binge drinkers for each emotion. There was weak evidence of an increased sensitivity towards happy faces in monthly compared to never/occasional binge drinkers ($p = .062$); there was no evidence of an effect of weekly binge drinking on happiness sensitivity compared to never/occasional binge drinkers ($ps = .720$). There was no evidence of an effect of monthly or weekly binge drinking compared to never/occasional drinkers on response sensitivity when seeing angry, sad, disgusted, fearful and surprised faces ($ps > .260$). Models were again adjusted for age, gender, education and trait anger (estimates in Table 3.3).

Table 3.3: Regression model estimates, 95 % Confidence intervals, and p-values for emotion specific response sensitivity (i.e., A' Prime) for each emotion.

	Anger			**Happy**			**Sad**			**Disgust**			**Fear**			**Surprise**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	.907	.769 – 1.045	**<0.001**	.901	.803 – .999	**<0.001**	.934	.849 – 1.020	**<0.001**	.955	.855 – 1.055	**<0.001**	.969	.653 – 1.286	**<0.001**	.905	.839 – .970	**<0.001**
Age	.0003	-.001 – .002	.614	.0001	-.001 – .001	.909	.0002	-.001 – .001	.660	-.001	-.002 – .0001	**.068**	-.002	-.005 – .001	.204	-.0003	-.001 – .0003	.283
Gender [Female= 0, Male= 1]	-.016	-.043 – .011	.239	-.019	-.038 – -.0002	**.048**	-.012	-.028 – .005	.172	-.020	-.040 – -.001	**.040**	-.081	-.143 – -.020	**.010**	-.016	-.029 – -.003	**.014**
Education [Degree or equivalent Higher education]	-.052	-.180 – .075	.419	-.016	-.107 – .075	.729	-.047	-.126 – .032	.243	-.024	-.117 – .068	.605	-.144	-.436 – .149	.334	-.003	-.063 – .057	.921
Education [A Level or equivalent]	-.039	-.170 – .092	.558	.009	-.084 – .102	.847	-.035	-.116 – .047	.402	-.029	-.124 – .066	.547	-.088	-.388 – .212	.565	-.003	-.065 – .059	.924
Education [GCSEs grades A*-C or equivalent]	-.040	-.174 – .094	.553	-.011	-.106 – .084	.821	-.041	-.124 – .042	.330	-.037	-.135 – .060	.448	-.059	-.366 – .248	.705	.015	-.049 – .078	.646
Education [Other qualifications]	-.043	-.188 – .102	.560	-.040	-.143 – .063	.447	-.037	-.127 – .053	.423	-.020	-.125 – .085	.711	-.137	-.470 – .196	.418	-.042	-.111 – .026	.226
Trait Anger	.001	-.002 – .003	.609	.00001	-.002 – .002	.988	.001	-.001 – .002	.305	-.0001	-.002 – .002	.881	-.002	-.007 – .003	.491	-.0001	-.001 – .001	.801
Binge Drinking [Monthly]	.001	-.029 – .030	.952	.020	-.001 – .041	**.062**	-.003	-.022 – .015	.727	.011	-.010 – .033	.302	.039	-.029 – .106	.260	.002	-.012 – .015	.830
Binge Drinking [Weekly]	.003	-.034 – .040	.892	.005	-.022 – .031	.720	-.003	-.026 – .020	.811	-.005	-.032 – .022	.708	.035	-.050 – .120	.415	-.002	-.020 – .015	.791
Observations		188			188			188			188			188			188	
R^2 / R^2 adjusted		.019 / -.031			.071 / .024			.033 / -.016			.052 / .004			.073 / .027			.074 / .027	

Notes: Age is years, *Gender [Female= 0, Male= 1], Education [Unknown/No Qualification =0,* Degree or equivalent Higher education =1, A Level or equivalent= 2, GCSEs grades A*-C or equivalent =3, Other qualifications =4], *Trait Anger Subscale of the STAXi-2, Binge Drinking [Never/Occasional = 0, Monthly = 1, Weekly =2]. A' Prime (Macmillan & Creelman, 2005; Pollack & Norman, 1964) is a measure of response sensitivity, and scores typically range from 0 (i.e., emotions cannot be recognised from noise) to 1 (i.e., emotions are perfectly distinguishable from noise.*

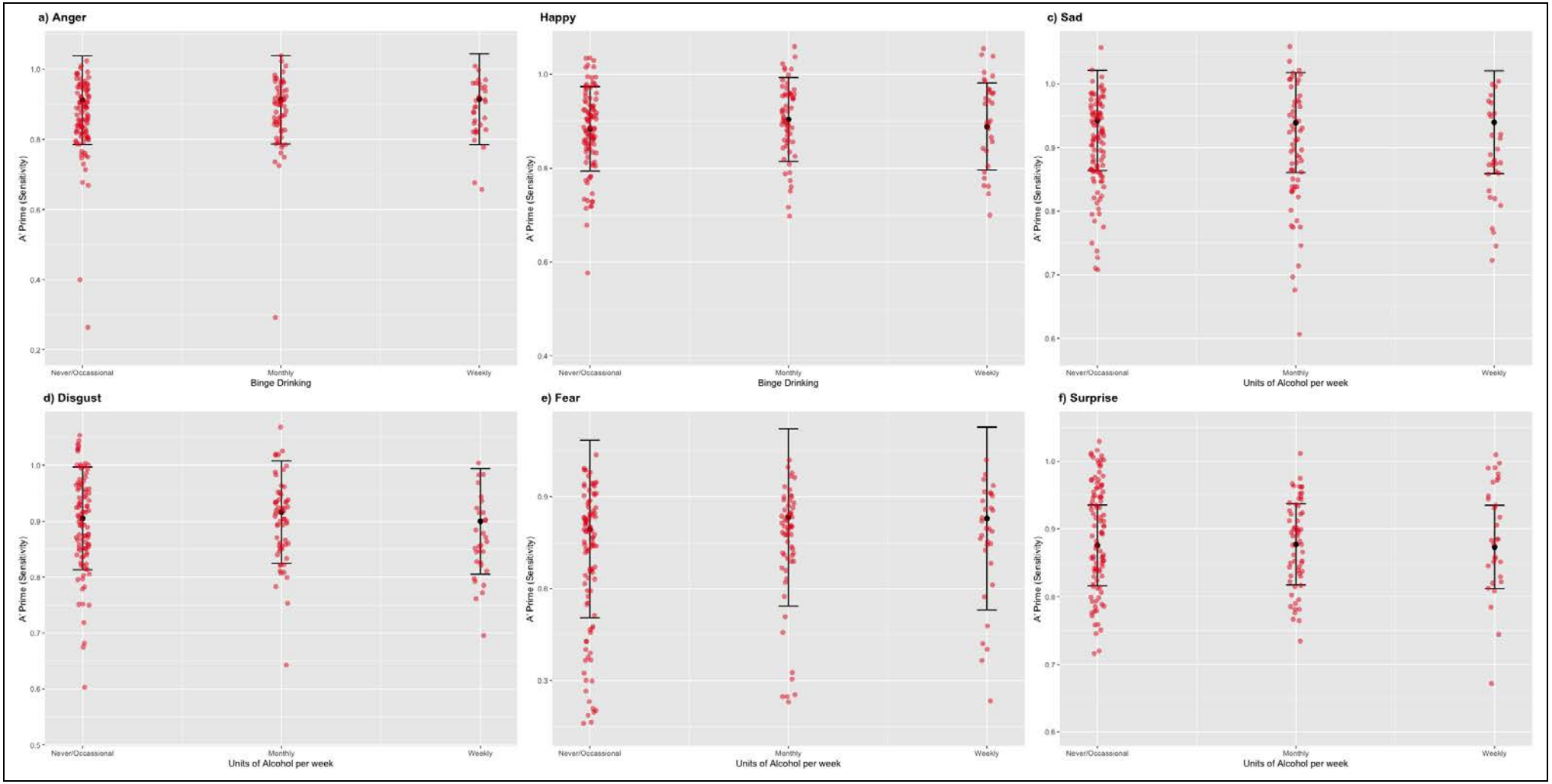

Figure 3.4: The association between binge drinking and emotion specific response sensitivity (i.e., A' Prime) for each emotion (Angry, Happy, Sad, Disgust, Fear, Surprise). Error bars are 95% confidence intervals.

3.5.4. Emotion Specific Response Bias

The model estimates of the association between chronic alcohol consumption and emotion specific response bias (i.e., B") for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise) can be found in Table 3.4. There was no association between units of alcohol consumed per week and emotion specific response bias for all emotions (*ps >.162*). Associations between units consumed per week and bias scores are displayed in Figure 3.5. Models were again adjusted for the influence of age, gender, education and trait anger. There was strong evidence for an association between age and response bias for disgusted emotions (*p <.001*) and weak evidence for an association for happy emotions (*p =.060*). Estimates suggest that as age increases individuals become less bias towards seeing disgusted faces and more bias towards seeing happy faces. There was weak evidence for an association between gender and response bias when seeing happy (*p =.057*) and sad faces (*p =.063*); estimates suggest that males are more bias towards seeing happiness and less bias towards seeing sadness. There was weak evidence for an association between education and response bias when seeing fearful faces. Estimates suggest that being educated to degree level or higher increases your bias towards seeing fear (*p =.067*) compared to having no qualifications. There was no evidence for an association between age, gender, education or trait anger and response bias for any of the remaining emotions (*p >.103*).

Table 3.4: Regression model estimates, 95 % Confidence intervals, and p-values for emotion specific response bias (i.e., B') for each emotion.

	Anger			**Happy**			**Sad**			**Disgust**			**Fear**			**Surprise**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	1.003	.669 – 1.338	**<0.001**	.517	-.184 – 1.219	.147	.357	-.224 – .938	.226	.070	-.517 – .657	.814	1.005	.565 – 1.445	**<0.001**	.379	-.063 – .821	.092
Age	-.0004	-.003 – .003	.807	-.006	-.013 – .0003	**.060**	-.002	-.007 – .004	.542	.010	.005 – .016	**<0.001**	-.002	-.006 – .002	.270	.001	-.003 – .005	.700
Gender [Female= 0, Male= 1]	-.037	-.102 – .028	.266	-.133	-.270 – .004	**.057**	.108	-.006 – .221	**.063**	.017	-.098 – .132	.769	-.060	-.146 – .026	.172	-.045	-.132 – .041	.301
Education [Degree or equivalent Higher education]	-.199	-.509 – .112	.208	.112	-.539 – .763	.735	-.008	-.547 – .532	.977	.194	-.352 – .739	.484	-.381	-.790 – .027	**.067**	-.131	-.541 – .279	.528
Education [A Level or equivalent]	-.128	-.446 – .190	.428	-.155	-.822 – .513	.648	-.041	-.595 – .512	.883	.267	-.292 – .827	.347	-.348	-.766 – .071	.103	-.115	-.535 – .306	.591
Education [GCSEs grades A*-C or equivalent]	-.174	-.501 – .152	.293	.248	-.437 – .932	.476	-.061	-.628 – .506	.832	-.006	-.579 – .568	.984	-.366	-.795 – .064	**.095**	.029	-.402 – .460	.895
Education [Other qualifications]	-.068	-.421 – .284	.703	.111	-.628 – .851	.767	-.048	-.661 – .565	.877	.163	-.457 – .782	.605	-.286	-.750 – .178	.226	-.106	-.572 – .360	.655
Trait Anger	-.003	-.009 – .003	.304	.008	-.004 – .019	.208	.005	-.005 – .014	.341	-.005	-.015 – .005	.305	-.001	-.008 – .006	.806	-.003	-.010 – .005	.444
Units of Alcohol per week	-.001	-.003 – .002	.587	-.002	-.007 – .003	.420	.002	-.002 – .006	.419	.0003	-.004 – .004	.876	.002	-.001 – .005	.162	.0005	-.003 – .003	.761
Observations		188			188			188			188			188			188	
R^2 / R^2 adjusted		.043 / -.0003			.092 / .051			.035 / -.009			.115 / .075			.047 / .004			.040 / -.003	

Notes: Age is years, Gender [Female= 0, Male= 1], Education [Unknown/No Qualification =0, Degree or equivalent Higher education =1, A Level or equivalent= 2, GCSEs grades A-C or equivalent =3, Other qualifications =4], Trait Anger Subscale of the STAXi-2, Units of Alcohol calculated from self-reported consumption data of standard drinks typically consumed per week. B" (Grier, 1971) is a measure of response bias. B" scores ranging from -1* (i.e., a response bias in favour of seeing the target emotion) to +1 (i.e., a response bias in favour of *not seeing the target emotion*)

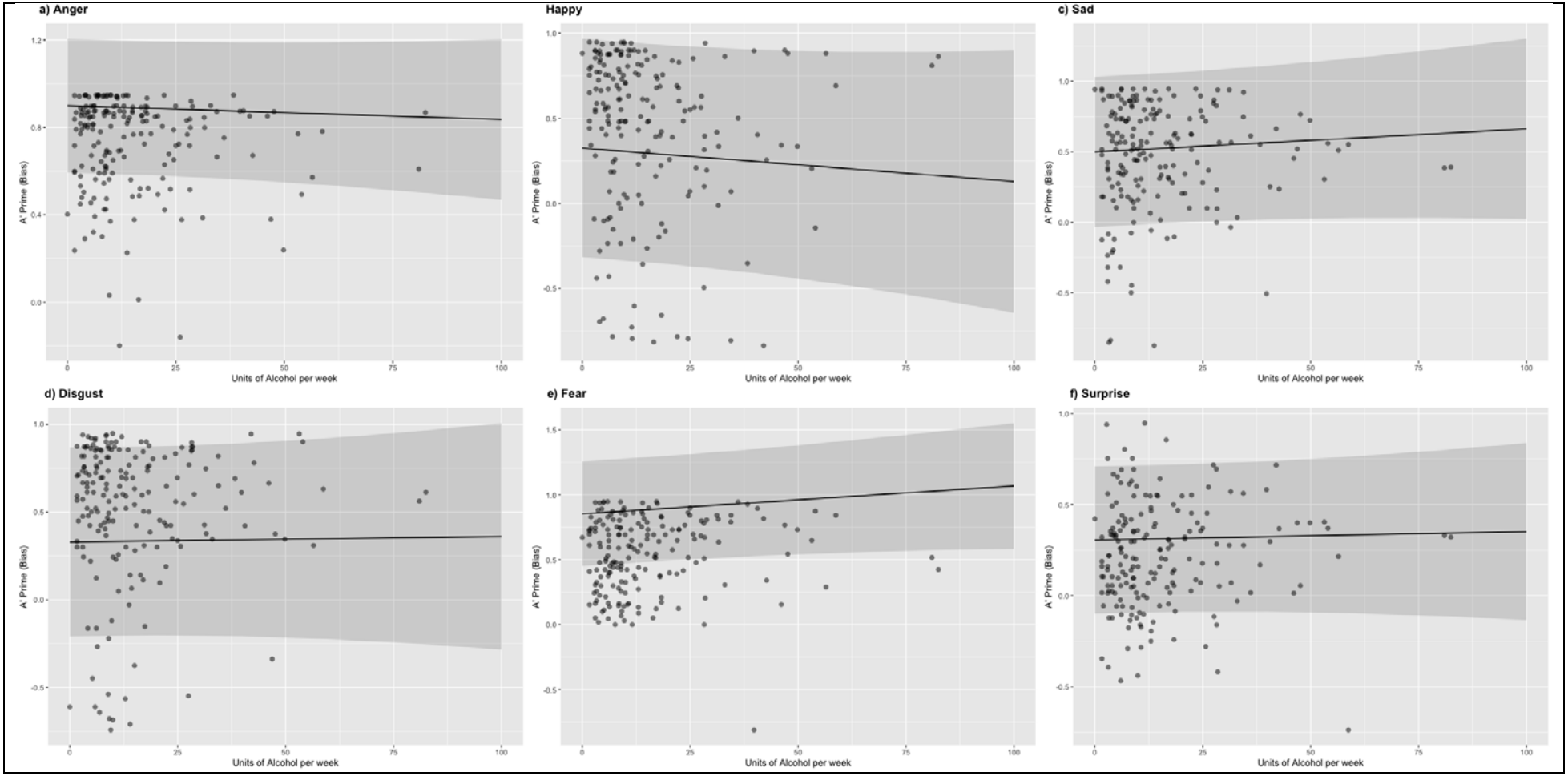

Figure 3.5: The association of units of alcohol per week with emotion specific response bias (i.e., B') for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise). Regression lines are estimated from each emotion model.

The model estimates of the association between binge drinking and emotion specific response bias (i.e., B") for each emotion (Anger, Happy, Sad, Disgust, Fear, Surprise) can be found in Table 3.5. Figure 3.6 displays observable differences between never, less than monthly, monthly and weekly binge drinkers for each emotion. There was no evidence of an effect of binge drinking (monthly or weekly frequency compared to never/occasional) on response bias when seeing angry, happy, sad, disgusted, fearful and surprised faces (*ps >.162)*. Models were again adjusted for age, gender, education and trait anger (estimates in Table 3.5).

Table 3.5: Regression model estimates, 95 % Confidence intervals, and p-values for emotion specific response bias (i.e., B") for each emotion.

	Anger			**Happy**			**Sad**			**Disgust**			**Fear**			**Surprise**		
Predictors	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*	*Estimates*	*CI*	*p*
(Intercept)	1.008	.671 – 1.345	**<0.001**	.565	-.140 – 1.269	.116	.356	-.231 – .942	.233	.126	-.461 – .712	.673	.961	.521 – 1.402	**<0.001**	.396	-.048 – .840	.080
Age	-.001	-.004 – .003	.744	-.006	-.013 – .0001	.053	-.001	-.007 – .004	.620	.011	.005 – .016	**<0.001**	-.002	-.006 – .002	.290	.001	-.003 – .005	.612
Gender [Female= 0, Male= 1]	-.037	-.102 – .029	.268	-.131	-.268 – .006	.061	.108	-.006 – .222	.062	.021	-.093 – .135	.716	-.061	-.147 – .024	.159	-.044	-.130 – .042	.316
Education [Degree or equivalent Higher education]	-.205	-.516 – .107	.197	.084	-.567 – .736	.799	.002	-.541 – .544	.996	.175	-.367 – .717	.525	-.357	-.764 – .051	.086	-.133	-.544 – .278	.524
Education [A Level or equivalent]	-.137	-.456 – .182	.398	-.186	-.853 – .482	.584	-.026	-.582 – .529	.925	.254	-.302 – .810	.369	-.323	-.741 – .095	.129	-.110	-.531 – .311	.607
Education [GCSEs grades A*-C or equivalent]	-.182	-.509 – .146	.275	.234	-.450 – .918	.501	-.047	-.616 – .523	.872	.001	-.569 – .570	.998	-.356	-.784 – .072	.102	.039	-.392 – .470	.859
Education [Other qualifications]	-.075	-.430 – .279	.675	.072	-.669 – .813	.848	-.039	-.655 – .578	.902	.130	-.487 – .746	.679	-.251	-.715 – .212	.287	-.111	-.578 – .356	.640
Trait Anger	-.003	-.009 – .003	.282	.007	-.005 – .019	.249	.005	-.005 – .015	.315	-.005	-.015 – .004	.273	-.0003	-.008 – .007	.937	-.003	-.010 – .005	.441
Binge Drinking [Monthly]	-.008	-.080 – .064	.824	-.100	-.251 – .050	.191	.004	-.121 – .129	.950	-.117	-.243 – .008	.066	.096	.001 – .190	**.047**	-.039	-.134 – .056	.421
Binge Drinking [Weekly]	.011	-.079 – .101	.810	-.021	-.210 – .168	.826	-.006	-.163 – .151	.942	-.034	-.191 – .123	.670	.051	-.067 – .169	.396	-.040	-.159 – .079	.510
Observations	188			188			188			188			188			188		
R^2 / R^2 adjusted	.042 / -.007			.097 / .052			.031 / -.018			.132 / .088			.058 / .010			.044 / -.004		

Notes: Age is years, *Gender [Female= 0, Male= 1], Education [Unknown/No Qualification =0,* Degree or equivalent Higher education =1, A Level or equivalent= 2, GCSEs grades A*-C or equivalent =3, Other qualifications =4], *Trait Anger Subscale of the STAXi-2, Binge Drinking [Never =0, Less than monthly =1, Monthly = 2, Weekly =3].. B" (Grier, 1971) is a measure of response bias. B" scores ranging from -1* (i.e., a response bias in favour of seeing the target emotion) to +1 (i.e., a response bias in favour of *not seeing the target emotion*)

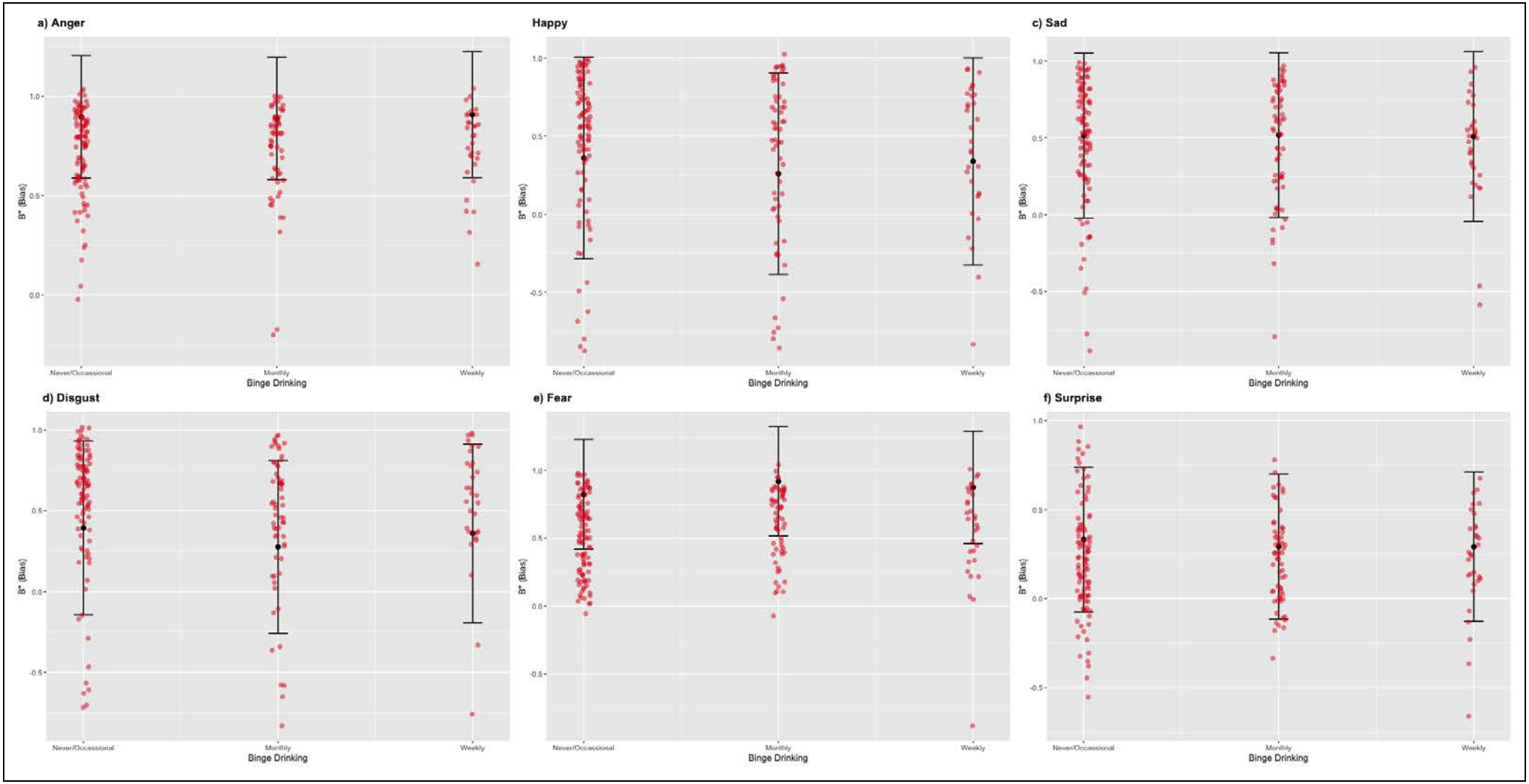

Figure 3.6: The association between binge drinking and emotion specific response bias (i.e., B") for each emotion (Angry, Happy, Sad, Disgust, Fear, Surprise). Error bars are 95% confidence intervals.

3.6. Discussion

This study aimed to test whether chronic alcohol consumption (i.e., units of alcohol consumed per week over a minimum period of 5 years) was associated with deficits in emotional face processing. It was anticipated that chronic consumption in non-dependent drinkers will result in poorer global emotion processing accuracy mimicking the deficit reported in alcohol dependent drinkers (Donadon & Osorio, 2017). Results however show no clear evidence of an association between units of alcohol consumed per week and global emotion processing accuracy (i.e., total hits) in a non-dependent sample. This suggests global deficits in recognition accuracy may only be present in alcohol dependent samples and the chronicity of alcohol consumption in non-dependent drinkers may not be sufficient enough to produce a deficit. This study also aimed to test association between binge drinking frequency and emotion processing. Similarly, global emotion processing accuracy did not differ between never/occasional, monthly and weekly binge drinkers. Again, it was anticipated that the increased frequency of this problematic drinking behaviour would result in a deficit in global emotion processing. These finding are echoed in a birth cohort longitudinal study testing late adolescent and early adulthood (i.e., ages 16-23) binge drinking behaviour on later emotion processing accuracy (i.e., age 24) (Mahedy et al., 2020). This study similarly reports no association between frequency of binge drinking and global emotion processing accuracy.

At an emotion specific level, it was anticipated that sadness perception specifically was likely to be influenced by chronic consumption as deficits have been reported in acute alcohol studies (Attwood, Ohlson, et al., 2009; Craig et al., 2009) as well as chronic studies testing alcoholic samples (Frigerio et al., 2002; Philippot et al., 1999). Results show modest evidence for a reduced sensitivity when seeing sad faces as units of alcohol consumed per week increases. This

finding is consistent with prior acute alcohol studies that show that non-dependent drinkers consuming alcohol within a single drinking session show similar sadness perception impairment (Attwood, Ohlson, et al., 2009; Craig et al., 2009). In chronic non-dependent drinkers, a reduced sensitivity towards sadness has key alcohol-related aggression implications. Sadness is typically considered to be a signal of distress or submission (Blair, 2005; Hart, 2011) and has the potential to curtail aggressive responding. Therefore, a reduced sensitivity towards seeing this emotion may be a mechanism increasing the likelihood of aggressive responding (as cues of distress and submission are likely to be missed). Result show no evidence of an association between units of alcohol consumed per week amongst non-dependent drinkers and anger, happy, disgust, fear and surprise response sensitivity. Associations between frequency of binge drinking and emotion specific response sensitivity was also tested. Results show modest evidence for an increase in happiness sensitivity amongst monthly binge drinkers compared to individuals that report never/occasionally binge drinking. This may be due to the context in which monthly binge drinking occurs. Given that alcohol is often consumed socially, this environmental exposure may influence the perception of positive emotions (i.e., happiness) which may explain the improves response sensitivity to this emotion in the present study. However, this must be interpreted with caution as there was no evidence to suggest that more frequent weekly binge drinkers differ in happiness sensitivity when compared to never binge drinkers. Associations between response bias for each emotion (angry, happy, sad, disgust, fear, surprise) and units of alcohol consumed per week were also tested. Prior research highlights that alcoholics tend to misidentify sad expressions as angry or disgusted facial expressions (i.e., demonstrating an anger/disgust bias). There was no evidence of an association between response bias and chronic consumption (i.e., units per week) for all emotional expressions. There was modest evidence to suggest that monthly binge drinkers have a

reduced bias towards fearful faces when compared to never/occasional binge drinkers. Again, these findings should be interpreted with caution as there was no clear evidence of an association between more frequent weekly binge drinkers and units of alcohol consumed per week.

This study is limited by cross-sectional design used. Longitudinal studies have the advantage of exploring the influence of alcohol consumption on emotional face processing amongst a non-dependent sample over time (Caruana et al., 2015). One advantage of this would be the reduced reliance on retrospective self-reported alcohol consumption typical in a cross-sectional investigation. Participants in this study were required to retrospectively report the number of alcoholic drinks consumed during a typical drinking week. From this information, the number of weekly UK units were estimated. However, evidence suggests a large discrepancy between what was actually consumed and what was reported (Gilligan et al., 2019) with heavy drinkers underestimating what they consume during a typical week (Gual et al., 2017). Recall bias resulting in inaccurate estimations of typical weekly alcohol consumption has led to the development of technology based momentary assessment of consumption behaviour whilst drinking occurs (Intille et al., 2016). This type of assessment gathers high temporal density longitudinal drinking habits data using a smartwatch that is low burden to the user. Future research aiming to capture chronic drinking patterns in non-dependent samples should aim to use such techniques. Longitudinal studies also have the distinct advantage of identifying and relating events to a particular exposure (I.e., alcohol consumption) and to further define these exposures with regards to chronicity (i.e., continued consumption over time) (Caruana et al., 2015). Future studies could build upon the results of the present study by investigating the influence chronic alcohol consumption has an emotional face processing using longitudinal data. Recent research has begun to investigate this using the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort (Mahedy et

al., 2020). These authors tested the association between chronic alcohol consumption and cognitive function. Binge drinking was assessed between the ages of 16-23 years old and cognitive measures of working memory, response inhibition and emotional face processing was assessed at age 24. They conclude that adolescent and early adulthood binge drinking was not associated with poorer cognitive function. Specifically, there was no clear association with global emotion processing deficits. Future studies should explore the influence of chronic consumption on emotion specific response sensitivity as well as response bias to investigate whether alcohol consumption over time is associated with impairments at an emotion specific rather than global only. Future research could also address the influence frequent alcohol consumption over time influences emotion processing in older participants. Specifically investigating early chronic alcohol exposure on later emotion processing deficits in older age groups.

Results from the present study suggest that chronic alcohol consumption (i.e., units consumed per week) and binge drinking frequency are not associated with anger perception sensitivity or response bias. Similarly, Chapter 2 suggests that acute alcohol consumption does not result in anger perception or response bias. Taken together, these results highlight that the perception of anger is not influenced by frequent drinking over time nor the acute exposure to alcohol. A possible for explanation for this surrounds the conceptual difference between hostile attribution bias and anger perception bias. Within the literature, an anger perception bias has been interpreted as a bias towards judging an expression as hostile (Smeijers et al., 2017). Conceptually however, ‘anger’ and ‘hostility’ differ (Eckhardt et al., 2004). Anger is referred to as an emotional state that conveys feelings including irritation, annoyance, fury and rage. The task used in the present study focuses on whether individuals can accurately recognise the emotional expression but does not require participants to evaluate the perceived emotional state or behavioural intentions.

Hostility on the other hand is an individual attitude that involves negative evaluations of others. Hostile interpretations of emotional stimuli may not only be towards angry faces alone. It is likely that other emotions, such as disgust or emotionally ambiguous facial expressions, may also be interpreted as hostile. This tendency to perceive or interpret others' behaviour as hostile is often referred to as hostile attribution bias (HAB) (Nasby et al., 1980). Research suggests that higher levels of this bias are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006). This can have negative social consequences, as perceived aggressive intent plays a causal role in reactive aggressive behaviour (Crick & Dodge, 1996).

Chapter 4 addresses facial expression interpretation, specifically whether acute alcohol consumption results in a great hostile attribution bias of emotional faces, rather than focusing on the accuracy of identifying the presence of a particular emotion in an expression. Recent research has investigated hostile attribution bias in facial affect using a sample of high aggressive individuals (i.e., forensic population) (Smeijers et al., 2017). These authors conclude that individuals with an aggression regulation deficit (i.e., antisocial and borderline personality disorder) demonstrate an increased perception of hostility in emotional expressions compared to healthy controls. Chapter 4 specifically tests whether acute alcohol consumption produces similar effects.

3.6.1. Conclusion

Findings seem to suggest an association between sadness specific deficits in emotion recognition rather than the anticipated global deficits. Longitudinal birth cohort studies have reported similar findings when assessing the influence of binge drinking on global emotion processing accuracy. The association found between increased chronic alcohol consumption (i.e., units of alcohol consumed per week) and a reduced sadness sensitivity is important considering

this emotion has been found to curtail aggression (i.e., cues distress and submission). Findings are however limited by the cross-sectional design used. Future research should assess associations between chronic alcohol consumption and emotional face processing longitudinally. This could involve using momentary assessment technology that collects high temporal density drinking data as the event occurs reducing recall bias. In addition, future work could investigate this topic using birth cohort data capturing drinking behaviour across a prolonged period of time. Chapter 4 focuses on investigating hostile attribution bias towards emotional stimuli with the aim of establishing whether aggressive behaviour following alcohol is due to deficits in emotion recognition or whether it is due to the interpretation of hostility when viewing facial expression.

CHAPTER 4: EFFECTS OF ACUTE ALCOHOL CONSUMPTION ON HOSTILE ATTRIBUTION BIAS TOWARDS EMOTIONAL FACIAL EXPRESSIONS AND IMPLICIT APPROACH/AVOIDANCE TENDENCIES

Keywords: Hostile attribution bias; emotional facial expressions; acute alcohol consumption; approach/avoidance tendency; alcohol related aggression.

4.1. Chapter Overview

The purpose of this chapter was to investigate whether social drinkers perceive emotional facial expression as more hostile following acute alcohol compared to placebo. Research suggests that aggressive individuals see emotional facial expressions as more hostile, compared to controls. Greater hostile attribution towards socially relevant cues has been found to increase aggression, and therefore could explain some instances of alcohol-related aggression. A further exploratory aim of this chapter was to investigate whether alcohol consumption influences the tendency to approach or avoid emotional facial expressions. Regular non-dependent drinkers (N=84) participated in a double-blind placebo-controlled experiment. Participants attended two sessions. In one, they consumed an alcoholic drink (0.4 g/kg), and in the other they consumed a matched placebo. In each session they completed two tasks. The first measured global and emotion specific hostile attribution bias towards emotional faces (happy, sad, angry, disgust, surprise, and fear). The second measured approach/avoidance tendencies towards emotional faces (angry, happy, sad and disgust). There was no evidence of a main effect of alcohol on global hostility ratings of emotional facial expression. However, there was evidence to suggest greater global hostile attribution bias of ambiguous emotional stimuli after alcohol (drink by intensity interaction). At an emotion specific level, happy faces were seen as more hostile after alcohol when compared to placebo (irrespective of emotional intensity). There was no evidence of greater approach/avoidance tendencies when seeing emotional faces following alcohol. These findings suggest that alcohol increases hostile judgements of ambiguous emotional faces. They also suggest that happy faces are perceived to be more hostile following alcohol. As an increased hostile attribution bias when processing socially relevant information increases aggressive responding, this increased hostile perception of happy faces following alcohol may increase the likelihood of aggressive behaviour.

4.2. Introduction

Emotional facial expressions are considered to be a fundamental component of effective social interaction (Moriya et al., 2013), which are capable of influencing behaviour. Research has demonstrated that acute alcohol consumption influences the perception of these emotional expressions. Our previous work suggests poorer emotion recognition following an acute dose of alcohol compared to placebo. At an emotion specific level, the ability to see sadness and fear is impaired (Eastwood et al., 2020). Attwood, Ataya, et al. (2009) reported an increased bias towards seeing anger in ambiguous facial morphs following acute alcohol consumption. This anger bias has been replicated in a more recent study, although effect sizes are small (Khouja et al., 2019). The reduced ability to see expressions associated with submission (i.e., sadness and fearful expressions) paired with an anger perception bias (albeit small), may function to increase aggressive responding. The tendency to perceive or interpret others' behaviour as hostile is often referred to as hostile attribution bias (Nasby et al., 1980). Research suggests that higher levels of this bias are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006). This can have negative social consequences, as perceived aggressive intent plays a causal role in reactive aggressive behaviour (Crick & Dodge, 1996).

Within the literature, an increased bias towards seeing anger has been interpreted as an increased bias towards judging facial expressions as hostile (Wegrzyn et al., 2017). However, 'anger' and 'hostility' conceptually differ (Eckhardt et al., 2004). Anger is an emotion most associated with feelings of irritation, annoyance, fury and rage. State-anger is often described as the response to an emotional elicitor that induces these feelings, whilst trait-anger is considered to be a more constant personality trait characterised by more frequent experiences of these feelings even when the cues are innocuous or unprovocative (Ramírez & Andreu, 2006). Hostility on the

other hand, can be considered to be an individual attitude that involves negative evaluations of others (Eckhardt et al., 2004). The perception of hostility communicates the intention to harm an individual, including expressive characteristics that signal intent for physical violence (Deffenbacher, 2000). In support of a difference between anger and hostility, one study found that facial displays of hostility produced greater physiological arousal than displays of anger (Tsikandilakis et al., 2020).

Hostile interpretations may not be restricted to angry faces. It is likely that other emotions, or emotionally ambiguous facial expressions, may also be interpreted as hostile. For example, a disgusted face in particular may be judged as more hostile as it shares similar expressive characteristics to anger (Wieser & Brosch, 2012). In addition, the unique characteristics of an emotional face, like disgust for example, may also contribute to hostile interpretations of these expressions (i.e., not just sharing similar expressive characteristics with anger, but displaying hostile expressive features in their own right). Recent research has investigated hostile attribution bias in facial affect using a sample of typically aggressive individuals (i.e., forensic outpatient population) (Smeijers et al., 2017). This research presented individuals with images of four facial expressions of emotion (angry, fear, disgust, and happy) which were judged as either displaying hostility or not. They found that individuals with an aggression regulation deficit (i.e., antisocial and borderline personality disorder) demonstrate an increased perception of hostility in emotional expressions (angry, disgusted, fearful and happy faces) compared to healthy controls. The authors discuss this hostile attribution bias towards emotional stimuli as a key characteristic of pathological aggression in a forensic outpatient sample. To our knowledge this is the first study to investigate hostile attribution bias of emotional stimuli by treating angry expressions and hostile judgements as separate concepts.

Another potential contributing factor in alcohol-induced aggression surrounds the behavioural response (i.e., whether to approach or avoid) when perceiving emotional facial expressions. Evidence typically suggests that we tend to approach positive stimuli and avoid negative stimuli (Chen & Bargh, 1999). In the facial affect literature, findings are inconsistent. One argument is that individuals tend to automatically avoid potentially threatening situations by approaching happy faces (Seidel et al., 2010), whilst avoiding angry faces (Heuer et al., 2007; Marsh et al., 2005). However, Veenstra et al. (2017) argue that individual differences in trait aggression influence these approach avoidance tendencies. They found that individuals high in trait aggression demonstrate quicker approach responses to angry faces. These finding could be attributed to increased testosterone levels associated with aggressive tendencies (Batrinos, 2012), biasing individuals to approach perceived surmountable social threat (Enter et al., 2014). This was further supported by Bossuyt et al. (2014) who concluded that approach/avoidance behaviour was goal-dependent; they found that angry faces were approached if the manipulated goal was to dominate/aggress. With regards to the alcohol literature, there is little research investigating approach/avoidance tendencies towards emotionally expressive facial stimuli under the influence of alcohol. Some evidence suggests that acute alcohol consumption increases testosterone levels when administered in low does (Sarkola & Eriksson, 2003; Sarkola et al., 2000). It is therefore anticipated that hostile cues (i.e., angry and disgusted faces) are less likely to be avoided and more likely to be approached following alcohol compared to placebo.

4.3. Aims

This research will investigate the effect of acute alcohol consumption on two primary outcomes. The first being hostile attribution bias of emotional facial expressions (happy, sad, angry, disgust, surprise, fear) and the second being approach/avoidance tendencies towards emotional

facial expressions (angry, happy, sad, disgust). An adapted version of the *Hostility Interpretation Bias Task* developed by Smeijers et al. (2017) will be used to measure hostile attribution bias in the present study. Participants will categorise composite images of happy, sad, angry, disgust, surprise and fearful male faces as either hostile or not hostile. We anticipate that emotional facial expressions will be seen as more hostile following alcohol consumption compared to placebo. The emotional intensity displayed in each face will range from ambiguous to full example along a 15 image continuum (consistent with previous facial expression research, Eastwood et al., 2020) as hostile attribution bias is particularly prevalent in ambiguous social context stimuli (Milich & Dodge, 1984). It was hypothesised that there will be greater hostile attribution bias towards emotional facial expression (i.e., increased percentage of hostile judgements) following acute alcohol consumption compared to placebo. In addition, emotion specific hostile attribution bias (i.e., % of hostile judgements) will also be explored following alcohol for each emotion (happy, sad, angry, disgust, surprise, fearful). For approach avoidance tendencies it is hypothesised that there will be less avoidance of hostile emotions (i.e., angry and disgusted) emotions (i.e., faster RTs in approach trials compared to avoidance) following acute alcohol compared to placebo. In addition, approach/avoidance tendencies (i.e., RTs) towards non-hostile emotions (i.e., happy and sad faces) will also be explored following acute alcohol, compared to placebo. As past research indicates greater hostile attribution bias in individuals that are typically aggressive (Smeijers et al., 2017) and demonstrate problematic drinking behaviour (Frigerio et al., 2002), trait aggression and hazardous drinking will be controlled for. Gender and age will also be measured to control for their influence.

4.4. Methods

4.4.1. Participants

Social drinkers (N = 84, 50% male) were recruited from the University of Bristol (staff and students) as well as the general population by means of existing email lists, poster advertisement and word of mouth. The inclusion criteria included: good physical and psychiatric health (self-report), aged between 18-40 and speak English as first language or equivalent level of fluency. To avoid including participants with little/no drinking experience or undiagnosed alcohol dependence, only individuals that consumed between 5 and 35 alcoholic units per week were included. One UK unit equals one 25 ml single measure of spirit (ABV 40%), or a third of a pint of beer (ABV 5-6%) or half a standard (175 ml) glass of red wine (ABV 12%) (NHS, 2018). The exclusion criteria were any individuals that reported a strong familial history of alcoholism (in parents and/or siblings) or that reported a history of psychiatric disorder (including drug addiction), alcohol consumption within 24 hours prior to testing or if their breath alcohol concentration (BrAC) was above zero (tested on arrival), and if they weighed less than 50kg if female or 60kg if male. Participants gave signed informed consent prior to taking part in the study. Participants were reimbursed £15 on completion of the study or were awarded equivalent course credits. The study was approved by the University of Bristol's Faculty of Science Human Research Ethics Committee (reference: 25011860401). The study protocol was pre-registered on the Open Science Framework (doi: 10.17605/OSF.IO/2EN6M).

4.4.2. Design

A double-blind placebo-controlled experimental design with one within subject factor of drink (0.4 g/kg alcohol, placebo) was used. The primary dependent measure of the HABT was percentage of 'hostile' responses. For this measure, an additional within-subjects factor of target

emotion was used (happy, sad, angry, disgust, surprise and fear). The primary dependent measure of the AAT was a RT bias score (i.e., approach RT scores subtracted from avoidance RT score). This measure included an additional within-subjects factor of emotion (happy, angry, sad, and disgust) and target face ethnicity (white, black). Error rate (i.e., proportion of errors) were also recorded to control for their influence on approach/avoidance RTs. Session order was counterbalanced with equal numbers of participants in each order group. Participants were allocated session orders in advance of the study using random number generator software (www.randomizer.org).

4.4.3. Drink

Drinks were prepared by a research collaborator who was independent of data collection and drink administration was double-blind. Alcohol content was dependent on participant weight. An upper limit of 90 kg was set so that participants weighing more than 90 kg received the same drink as a 90 kg participant. The alcoholic drinks were mixed using one-part vodka (37.5% ABV) to three parts tonic water. The dose used was 0.4 grams of alcohol per kg of body weight (g/kg (Attwood, Ataya, et al., 2009; Craig et al., 2009). Placebo drinks were matched volume tonic water. In order to mask the taste of alcohol, drinks were chilled and flavoured with lime cordial (40 ml) prior to serving. The inside rim of the glass was sprayed twice with a vodka mist.

4.4.4. Materials

4.4.4.1. Hostile Attribution Bias Task (HABT)

The HABT was an adapted version of the Hostility Interpretation Bias Task (HIBT) developed by (Smeijers et al., 2017). In the HABT, the images used were composite (i.e., prototypical) images created from photographs of 12 young male adults photographed under controlled conditions. Each trial began with a centrally-displayed fixation cross. A 350 × 457 pixel

face stimulus was then presented for 150 ms, followed by a noise mask for 250 ms in order to prevent after-image effects. The HABT was run using E-Prime 2.0 Pro software, on a standard computer with a QWERTY keyboard. Six 15-image morph sequences were created, one for each emotion (happy, sad, angry, disgust, surprise and fear). These run along a linear continuum from a neutral (i.e., emotionally ambiguous) prototype to the full emotional intensity (i.e., emotionally unambiguous). On each trial, a single image from the 90 available was presented for 150ms (backward masked). Labels were displayed on the bottom left (Hostile) and bottom right (Not Hostile) of the screen in *Arial*, size 30 font. Each image was presented twice, giving 180 trials in total. Participants were required to identify whether the image looked hostile using the 'c' key or 'not-hostile' using the 'm' key as quickly as possible. The outcome measure of interest was the percentage of hostile responses.

4.4.4.2. Approach avoidance task (AAT)

This task used the similar face stimuli as described in the HABT. However, only full intensity stimuli of angry, happy, sad and disgusted faces were used. The task consists of a practise block of 12 trials using neutral facial expression images, followed by 2 experimental blocks, each comprising 64 trials (i.e., 128 trials in total). Each experimental block uses white and black ethnicity examples of angry, happy, sad and disgusted faces repeated four times (i.e., 32 images). Each of these stimuli were presented as an approach and an avoidance trial (i.e. 64 images in total per block). On each trial, a fixation cross appears on screen for 500 milliseconds (ms), before being replaced by an image (i.e., face stimuli). After a short delay (500 -750 ms), a solid line or dashed black frame appeared around the image. A solid line frame cued the participant to approach by pulling the image towards them (arm flexion; 50% of trials), using a computer joystick and doing so increased the size of the image. A dashed black frame cued the participant to avoid by pushing

the image away from them (arm extension; 50% of trials) and doing so reduced the size of the image (Phaf et al., 2014). This zooming effect provided the participant with feedback on each trial so that the image increasing and decreasing in size reinforced a sense of approach and avoidance, respectively (Phaf et al., 2014). The stimulus remained on the screen for 10,000ms and participants were encouraged to respond as quickly and as accurately as possible. If no response was generated within this allotted timeframe the trial was over. The order of stimuli presentation was randomised within the blocks and across participants. The primary measures of interest were approach/avoidance RTs and error rates. Consistent with previous research involving the AAT, median RTs were used as they are less sensitive to extreme outliers (Wiers et al., 2011). Each median RT was measured from the presentation of the cue to the disappearance of the image (following a response) in the approach and avoidance trials. Error rates were proportion of errors made (i.e., approach response in an avoidance cued trial/avoidance response in an approach cued trial).

4.4.4.3. Questionnaires

Questionnaires used were the Alcohol Use Disorders Identification Test (AUDIT) (Saunders et al., 1993); higher scores on this measure indicate more problematic drinking behaviour consumption. The State anger (S-Ang) and Trait anger (T-Ang) subscales of the State-Trait Anger Expression Inventory (STAXI-2) (Spielberger, 1999); higher scores on each subscale represent great aggressive tendencies. The Positive and Negative Affect Schedule (PANAS) (Watson et al., 1988); higher scores represent greater positive/negative affect.

4.4.5. Procedures

Eligible participants were required to attend two testing sessions (approximately 60 minutes each), scheduled at least 7 days apart. In session one, participants were given the

opportunity to read the information sheet again and ask questions, before providing written informed consent. The researcher then conducted a short screening procedure to verify eligibility, which included measures of weight and an alcohol breath test (Draeger AlcoDigital 3000 Breathalyzer) to confirm zero breath alcohol concentration (BrAC). Weight information was passed to a research collaborator for drink preparation. Participants then completed baseline questionnaire measures (pre-consumption), including the AUDIT, STAXI-2, and PANAS. During both testing sessions, participants received a single drink to consume. In session one, this contained either 0.4 g/kg alcohol or a matched placebo. The opposite drink was administered in session two (order counterbalanced). Participants were given 10 minutes to consume all of their drink and a further 10 minutes to sit quietly to allow for absorption. Next, participants completed the HABT and the AAT (fixed order). They then completed the questionnaire measures a second time post-consumption (STAXI-2; State Anger Subscale (S-Ang), PANAS), and a second BrAC reading. Before leaving the session, participants were required to read and sign a safety card and were offered the opportunity to stay behind until they feel any effects of alcohol have worn off. They were also offered a taxi home if required. At the end of session two, participants were debriefed and reimbursed.

4.4.6. Sample Size Calculation

Sample size was determined from an effect size obtained in our previous study that investigated the effects of an acute dose of alcohol on emotional facial expression processing, in high vs low trait aggressive drinkers (Eastwood et al., 2020). This study used a 6-alternative forced choice task (6AFC) to investigate global emotion processing accuracy, using total hit rate as the primary outcome measure, and indicated that alcohol administration resulted in lower emotion processing accuracy (M=99.80, SD = 12.61) compared to placebo (M = 103.37, SD = 12.37). This

suggested an effect size of *dz* = .36 (correlation between conditions r = .68). Based on these data, we would need a total sample size of 84 participants to achieve 90% power (alpha level of 5%) to observe an effect of alcohol on hostile attributions of a similar size. Interaction analyses will be exploratory.

4.4.7. Statistical Analysis

Statistical analyses were conducted using R Studio (2019) (r version: R Core Team). For the HABT data, an error in programming the task meant that the presentation of the surprise emotion was compromised. This error meant that some of the participants were presented with two full intensity surprise images and 28 emotionally ambiguous images (i.e., 5% along the continuum between 'emotional ambiguity' to 'full intensity' surprise) when completing the task. As a result, the erroneous surprise data was removed from analysis. It was originally planned that this data would be analysed using 2 drink (alcohol, placebo) x 5 emotion (happy, sad, angry, fear, disgust) repeated measures ANOVA with interactions being explored using t-tests. However, it was later decided that this data would be analysed using linear mixed effects (LME) modelling (Baayen et al., 2008). This allows for the systematic control over the random between-subject's variance whilst controlling for other fixed effect variance (age, gender, trait anger, AUDIT). It also allows for the exploration of the interaction between emotion intensity (i.e., [1] emotionally ambiguous – [15] full emotionally intensity) and Drink (alcohol, placebo). Multivariate normality, homoscedasticity and multicollinearity assumptions were satisfied unless otherwise stated. The primary objective of this research was to investigate whether alcohol influences Hostile Attribution Bias when perceiving emotional facial expressions and this study was powered to detect an effect of drink (i.e., alcohol vs placebo). To test hostile ratings of emotional facial expressions globally, LME models were used with two blocks (lme4 package in r; Bates et al., 2015). In the first block

(*main effects block*), fixed effects of drink and intensity were entered into the LME model to test the main effects of drink and intensity on hostile ratings. In the second block (*interaction block*), a drink by intensity interaction term was entered into the model to test the interaction between drink and emotional intensity on hostility ratings. Age, gender, trait anger and AUDIT scores were also entered as fixed effects to adjust for their influence. Random intercepts for subject ID, as well as by ID random slopes for the effect of drink were entered as random effects. P values for each fixed effect were estimated using Kenward-Roger degrees of freedom (Luke, 2017). For emotion specific analyses of angry, happy, sad, disgust and fearful faces the same LMM models were applied.

For the AAT, 3 participants were removed from the analysis as error rates were relatively high (3 standard deviations from the mean). Further inspection of these data suggests that the participants misunderstood the task (i.e., high errors scores indicated using the task response options the wrong we around). It was planned that this data would be analysed using a 2 drink (alcohol, placebo) x 2 ethnicity (white, black) x 4 emotion (happy, angry, sad, disgust) repeated measures ANOVA to explore RT bias scores (i.e., the difference between approach and avoidance RT scores). It was also planned to analyse error rates using a 2 drink (alcohol, placebo) x 2 ethnicity (white, black) x 4 emotion (happy, angry, sad, disgust) x 2 tendency (approach, avoidance) repeated measures ANOVA. Instead, LME models were used to investigate approach/avoidance tendencies (i.e., approach and avoidance RTs) following alcohol and placebo, for each of the 4 emotions (happy, angry, sad, disgust). For each emotion, LME models were used with two blocks (lme4 package in r; Bates et al., 2015). In the first block (*main effects block*), drink and tendency were entered into the model as a fixed effect. In the second block (*interaction block*), a drink by tendency interaction term was entered into the model. Error rate was also

entered as a fixed effect to control for their influence, as well as age, gender, trait anger, AUDIT scores, face ethnicity. As random effects, we had random intercepts for subject ID, as well as by ID random slopes for the effect of drink. P values for each fixed effect were estimated using Kenward-Roger degrees of freedom (Luke, 2017).

4.5. Results

4.5.1. Participant Characteristics

Participant were aged between from 18-40 (M= 22.5, SD= 4.39) and weighed between 50-117kg (M= 71.24, SD= 13.03). Scores on the AUDIT ranged from 3-34 (M= 10.3, SD= 4.86) and scores on the trait aggressive subscale of the STAXI-2 ranged from 10-28 (M= 15.60, SD= 3.72). When asked on completion of each testing session, 83.3% of participants believed they had consumed alcohol when the drink administered was alcohol. In comparisons, 40.5% believed they had consumed alcohol when the drink administered was placebo.

4.5.2. Hostile Attribution Bias of Emotional Stimuli

4.5.2.1. Global Emotion Hostile Ratings

Mean % of hostility rating scores (SD) are displayed in Table 4.1 for each emotion individually as well as the global rating score. Table 4.2 displays the block 1 (main effects) and block 2 (interaction effects) model estimates for the Hostile Attribution Bias towards emotional facial expressions. Block 1 shows that there was no evidence for a main effect of drink on hostile ratings of facial expressions ($p = .342$) but there was strong evidence for a main effect of intensity ($p < .001$). Faces were rated an estimated 1.6% more hostile as emotionally intensity incrementally increased. Block 2 shows modest evidence for a drink by intensity interaction ($p = .002$; *see* Figure 4.2a) Estimates suggest a 3.5% increase in hostility rating following alcohol when the emotional intensity displayed was 0 (i.e., emotionally ambiguous). Following alcohol, as the intensity of the emotion displayed increased, hostility ratings increased by an estimated 1.5% for every incremental step (0 emotionally ambiguous - 15 full emotional intensity). Following placebo, as the intensity of the displayed emotion increased, hostility ratings increased by an estimated 1.8% for every incremental step (Figure 4.2a). The LME model adjusted for the influence of age, gender,

trait aggression, AUDIT score. There was evidence for a decrease in Hostile Attribution Bias as age increased (*p =.002*) and as AUDIT scores increased (*p =.036*). There was no evidence of a gender or trait anger effect (*ps >.564*).

Table 4.1: Mean and standard deviation (SD) for Hostility ratings of emotional facial expression (Angry, Happy, Sad, Disgust, Fear) following alcohol and placebo drinks.

Emotion	**Alcohol**		**Placebo**	
	Mean	**SD**	**Mean**	**SD**
Angry	71.76	17.67	72.11	15.62
Happy	8.09	12.87	5.07	10.24
Sad	10.10	16.40	8.62	15.20
Disgust	63.58	30.41	60.74	29.82
Fear	4.52	12.42	6.03	18.68
Global	**31.61**	**17.95**	**30.51**	**17.91**

Notes: n=84

4.5.2.1. Emotion Specific Hostile Ratings

The block 1 (main effects) and block 2 (interaction effects) model estimates of hostile attribution bias (i.e., % of hostile responses) for each emotion (Angry, Happy, Sad, Disgust, Fear) are displayed in Table 4.2. Block 1 shows that there was evidence for an increase in hostility ratings of happy emotions following alcohol compared to placebo (*p =.009;* see Figure 4.1); ratings were an estimated 3.0% higher following alcohol. There was no evidence for a main effect of drink on hostile ratings of angry, sad, disgusted and fearful emotional expression (*ps >.211*). There was

strong evidence for a main effect of intensity on Hostile Attribution Bias of angry, happy, and disgusted faces (*ps <.001*). Hostility rating of angry and disgusted emotion increased an estimated 5.5% and 3.3% (respectively) for every unit increase in emotional intensity. For happy faces, hostility ratings decreased an estimated .6% for every unit increase in intensity. There was modest evidence for an effect of intensity on hostility ratings of sad expression (*p =.006*); estimates suggest a .2% decrease in ratings for every unit increase in emotional intensity. There was no evidence for a main effect of intensity for fearful emotions (*p =.741*). Block 2 shows modest evidence for a drink by intensity interaction on hostile ratings of angry emotions (*p =.019;* see Figure 4.2*b*). Estimates suggest a 4.4% increase in hostility rating of angry expressions following alcohol when the emotional intensity was 0 (i.e., emotionally ambiguous). Following alcohol and as the intensity of anger displayed increased, hostility ratings increased an estimated 5.2% for every incremental step. Following placebo and as the intensity of anger displayed increased, hostility ratings increased an estimated 5.8% for every incremental step. There was also weak evidence for an interaction effect on hostile ratings of disgusted faces (*p =.059;* see Figure 4.2e). Estimates suggest a 6.7% increase in hostility rating of disgusted expressions following alcohol when the emotional intensity was 0 (i.e., emotionally ambiguous). Following alcohol and as the intensity of disgust displayed increased, hostility ratings increased an estimated 3.1% for every incremental step. Following placebo and hostile ratings increased an estimated 3.5% for every incremental increase in intensity. There was no evidence of an interaction for happy, sad and fearful expressions (*ps >.131*). Each LME model controlled for the influence of age, gender, trait anger, AUDIT score. There was evidence to suggest that hostile judgement decreased as age increased for angry, happy and disgusted faces (*ps< .035*) but not for sad and fearful faces (*ps> .288*). There was no evidence of an effect of gender on hostile judgements of all emotions (*ps> .421*). There

was modest evidence of an increase in hostile judgements of angry faces as trait level of aggression increases ($p = .022$) but not for happy, sad, disgusted and fearful faces ($ps > .225$). There was evidence of a decrease in hostile judgements of angry and disgusted faces as AUDIT score increased (i.e., an increase in hazardous drinking) ($ps < .049$), but not for happy, sad or fearful faces ($ps > .154$).

Table 4.2: LME model main effect estimates, 95 % Confidence intervals, and p-values for global hostile ratings of emotional faces, as well as for each specific emotion (Angry, Happy, Sad, Disgust, Fear). Random effect variance for subject ID and Drink.

Fixed Effects

	Global			**Angry**			**Happy**			**Sad**			**Disgust**			**Fear**		
Predictors	*Estimate*	*95% CI*	*p*	*Estimate*	*95% CI*	*p*	*Estimate*	*95% CI*	*p*	*Estimate*	*95% CI*	*p*	*Estimate*	*95% CI*	*p*	*Estimate*	*95% CI*	*p*
Block 1: Main Effects (Intensity + Drink)																		
(Intercept)	43.45	27.70 – 59.19	**<0.001**	50.960	31.806 – 70.115	**<0.001**	18.627	4.656 – 32.599	**0.010**	23.530	2.676 – 44.383	**0.027**	115.640	79.525 – 151.755	**<0.001**	10.913	-7.204 – 29.030	0.234
Age	-0.89	-1.46 – -0.33	**0.002**	-1.382	-2.067 – -0.697	**<0.001**	-0.541	-1.043 – -0.040	**0.035**	-0.402	-1.150 – 0.346	0.288	-2.292	-3.589 – -0.995	**0.001**	-0.076	-0.719 – 0.566	0.814
Gender [Female [R], Male]	-1.46	-6.47 – 3.55	0.564	-1.567	-7.635 – 4.501	0.609	1.196	-3.244 – 5.635	0.593	-2.693	-9.318 – 3.933	0.421	-3.952	-15.438 – 7.534	0.495	-1.794	-7.487 – 3.898	0.532
Trait Anger	0.03	-0.65 – 0.72	0.919	0.972	0.141 – 1.803	**0.022**	0.373	-0.235 – 0.981	0.225	-0.017	-0.924 – 0.890	0.971	-0.859	-2.432 – 0.713	0.280	-0.065	-0.845 – 0.714	0.868
AUDIT	-0.54	-1.05 – -0.04	**0.036**	-0.613	-1.224 – -0.002	**0.049**	-0.324	-0.771 – 0.124	0.154	-0.214	-0.882 – 0.453	0.525	-1.397	-2.555 – -0.240	**0.019**	-0.134	-0.708 – 0.439	0.642
Intensity	1.61	1.51 – 1.71	**<0.001**	5.513	5.264 – 5.762	**<0.001**	-0.561	-0.705 – -0.417	**<0.001**	-0.242	-0.412 – -0.071	**0.006**	3.297	3.052 – 3.542	**<0.001**	0.017	-0.083 – 0.116	0.741
Drink [Placebo [R], Alcohol]	1.06	-1.15 – 3.28	0.342	-0.360	-3.620 – 2.900	0.827	3.013	0.759 – 5.268	**0.009**	1.388	-1.539 – 4.316	0.348	2.937	-1.698 – 7.571	0.211	-1.536	-4.957 – 1.885	0.375
Block 2: Interaction Term (Intensity * Drink)																		
(Intercept)	42.24	26.48 – 58.00	**<0.001**	48.590	29.339 – 67.841	**<0.001**	18.316	4.298 – 32.334	**0.011**	22.756	1.860 – 43.653	**0.033**	113.752	77.585 – 149.919	**<0.001**	10.299	-7.836 – 28.433	0.262
Intensity	1.76	1.62 – 1.90	**<0.001**	5.810	5.459 – 6.162	**<0.001**	-0.522	-0.726 – -0.318	**<0.001**	-0.145	-0.387 – 0.097	0.240	3.533	3.187 – 3.880	**<0.001**	0.094	-0.047 – 0.234	0.193
Drink [Placebo [R], Alcohol]	3.48	0.78 – 6.19	**0.012**	4.399	-0.725 – 9.524	**0.092**	3.635	0.423 – 6.847	**0.027**	2.932	-1.058 – 6.922	0.149	6.712	0.665 – 12.759	**0.030**	-0.310	-4.074 – 3.454	0.871
Intensity * Drink [Placebo [R], Alcohol]	-0.30	-0.50 – -0.11	**0.002**	-0.595	-1.092 – -0.098	**0.019**	-0.078	-0.366 – 0.210	0.597	-0.193	-0.535 – 0.149	0.268	-0.472	-0.962 – 0.018	**0.059**	-0.153	-0.352 – 0.046	0.131

Table continues on next page.

	Global		Angry		Happy		Sad		Disgust		Fear	
Random Effects												
	Variance	*SD*	*Variance*	*SD*	*Variance*	*SD*	*Variance*	*SD*	*Variance*	*SD*	*Variance*	*SD*
Residual	117.29	10.830	754.9	27.48	253.29	15.915	356.4	18.88	734.5	27.10	120.8	10.99
ID	128.27	11.326	151.2	12.30	82.63	9.090	223.2	14.94	671.0	25.90	352.6	18.78
Drink [Placebo, Alcohol]	88.71	9.418	124.9	11.18	74.08	8.607	134.4	11.59	358.1	18.92	232.4	15.24
ICC	0.54		0.20		0.31		0.40		0.49		0.68	
N	84 $_{ID}$		84 $_{ID}$		84 $_{ID}$		84 $_{ID}$		84 $_{ID}$		84 $_{ID}$	
Observations	2520		2517		2517		2517		2520		2512	
Marginal R^2 / Conditional R^2	0.218 / 0.644		0.395 / 0.517		0.043 / 0.340		0.014 / 0.404		0.213 / 0.598		0.007 / 0.677	

Note: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Intensity of emotion (1 – emotionally ambiguous – 15 full example of emotion), Drink (Placebo, Alcohol). LME models were used to explore global as well as emotion specific hostile attribution bias. In Block 1, the fixed effects of drink and intensity were entered into each linear mixed effect model to test the main effects of drink and intensity. In Block 2, a drink by intensity interaction term was entered into each model. Age, gender, trait anger and AUDIT were also entered into each model to control for their influence. Random effects for each full model included random intercepts for subject ID and by ID random slopes for the effect of drink. P values for each fixed effect were estimated using Kenward-Rogers d.f.
^{R} Reference level for each categorical variable.

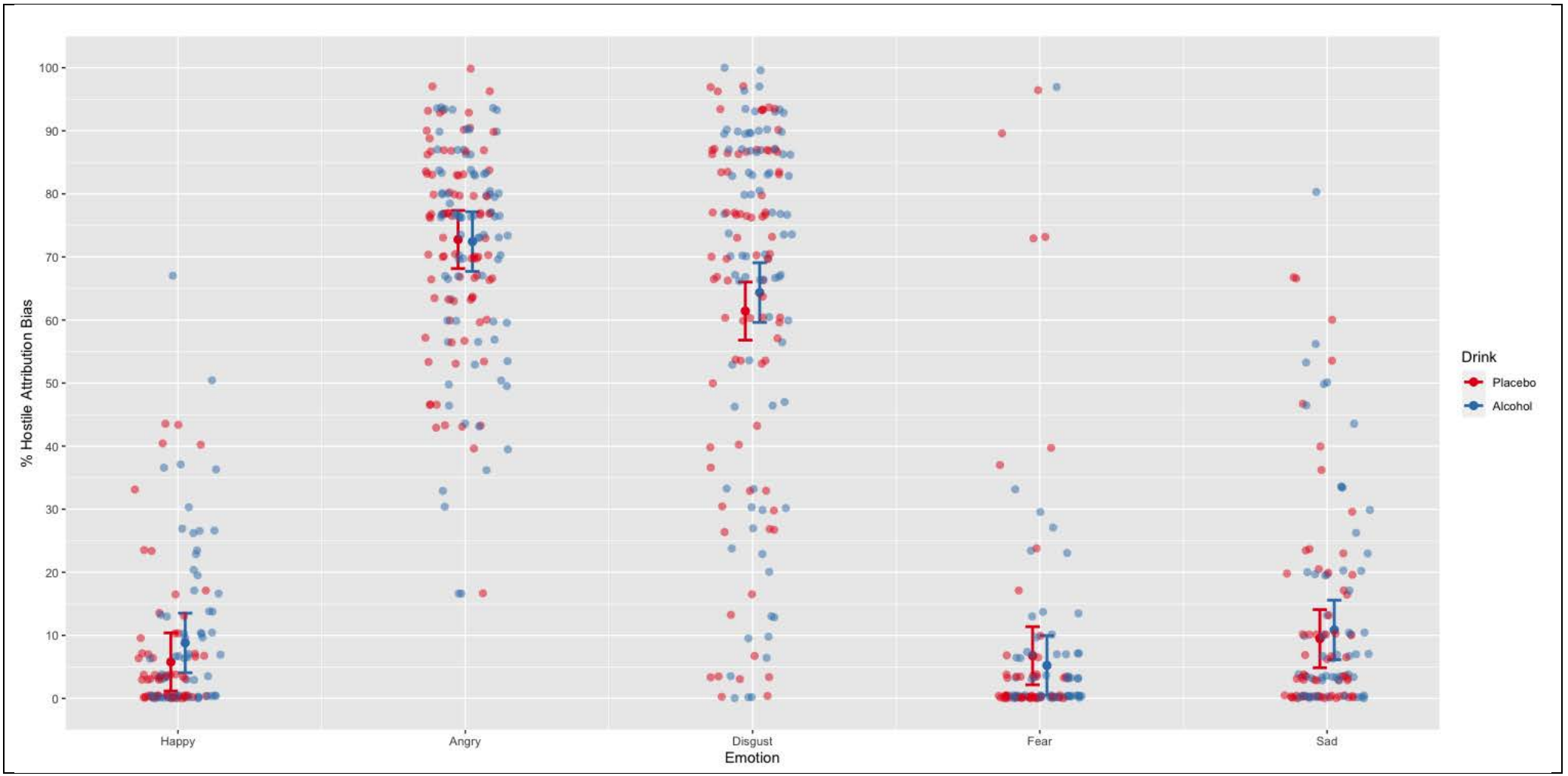

Figure 4.1: % Hostility rating for each emotional facial expression (Happy, Angry, Disgust, Fear, Sad) following alcohol and placebo drinks. Error bars represent 95% confidence intervals.

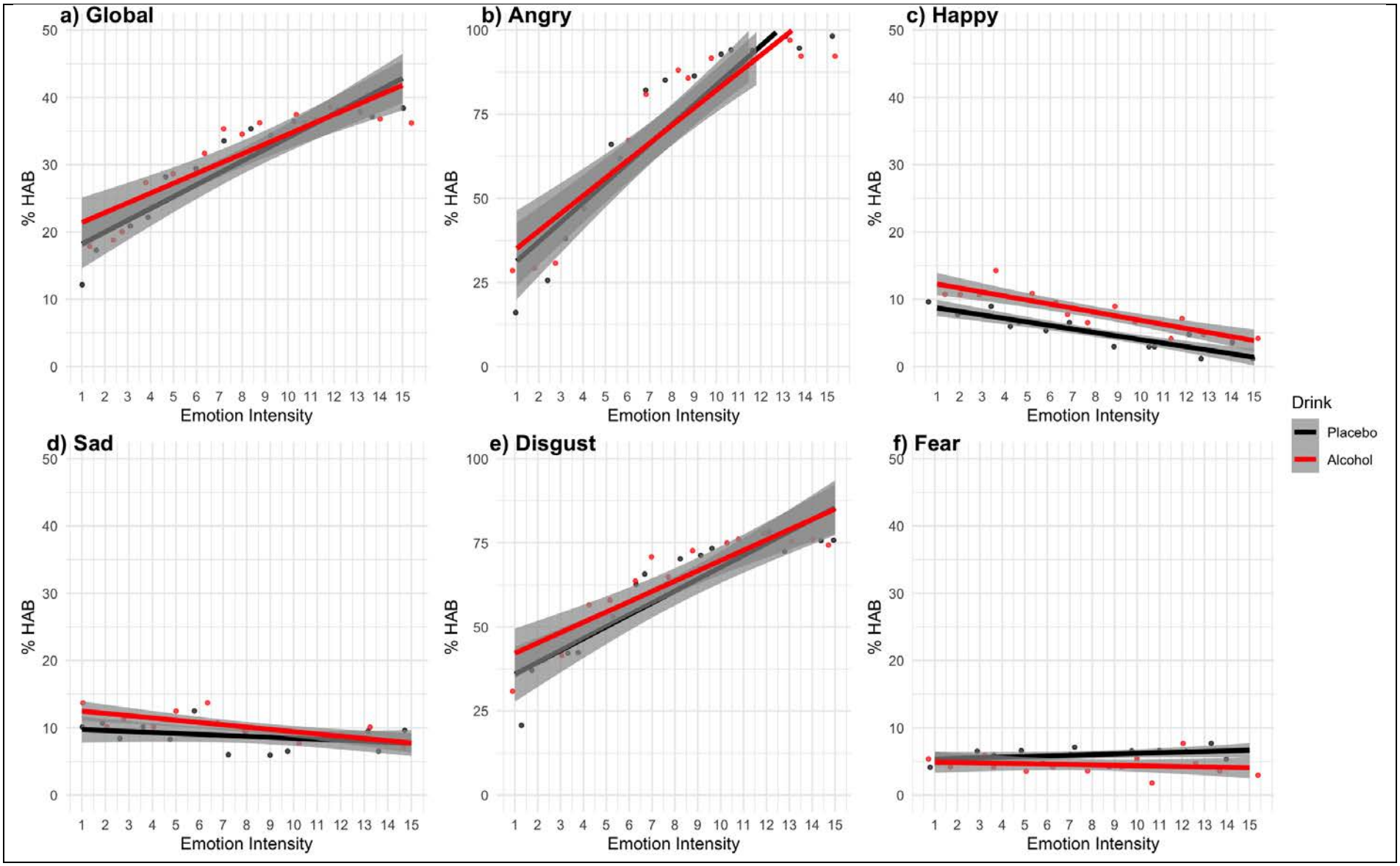

Figure 4.2: The relationship between % Hostile Rating and emotional intensity (1 – emotionally ambiguous – 15 full example of emotion) following alcohol and placebo drinks. a) Global hostile ratings of emotional facial expressions, b) hostile ratings of angry face.

4.5.3. Approach/Avoidance of Emotional Stimuli

The block 1 (main effects) and block 2 (interaction effects) model estimates of approach/avoidance RT bias score for each emotion (Angry, Happy, Sad, Disgust) are displayed in Table 4.3. Block 1 shows that there was modest evidence for a main effect of drink for each emotion (ps <.007). For angry, happy, sad and disgusted facial expression, RTs were an estimated 21.8ms, 26.0ms, 20.6ms and 26.0ms faster following alcohol compared to placebo, respectively. There was also strong evidence for a main effect of tendency for each emotion (*ps <.002*). For angry, happy, sad and disgusted facial expression, RTs were an estimated 26.7ms, 19.7ms, 16.4ms and 13.2ms faster in the approach compared to avoid cued condition, respectively. Block 2 shows no evidence of a tendency by drink interaction on RTs for each emotion (*ps >.268;* see Figure 4.3). Each LME model controlled for the influence of age, gender, trait anger, AUDIT score, face ethnicity, and AAT error rate. There was modest evidence to suggest that males responded faster to all emotional faces than females (*ps <.007*). There was no evidence for an effect of age, trait anger, AUDIT score, face ethnicity, or error rate on RT (*ps >.298*).

Table 4.3: LME model estimates, 95 % Confidence intervals, and p-values for approach/avoidance RT scores for each specific emotional facial expression (Angry, Happy, Sad, Disgust, Fear). Random effect variance for subject ID and Drink.

Fixed Effects

	Anger			**Happy**			**Sad**			**Disgust**		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
Block 1: Main Effects (Tendency + Drink)												
(Intercept)	685.307	542.796 – 827.819	**<0.001**	675.494	545.993 – 804.995	**<0.001**	669.927	533.348 – 806.506	**<0.001**	684.945	545.912 – 823.978	**<0.001**
Age	-0.793	-6.184 – 4.599	0.770	0.331	-4.557 – 5.219	0.893	0.156	-5.016 – 5.327	0.952	-0.330	-5.591 – 4.931	0.901
Gender [Female [R], Male]	-61.118	-105.226 – -17.010	**0.007**	-53.081	-93.046 – -13.116	**0.010**	-52.461	-94.759 – -10.162	**0.016**	-56.529	-99.567 – -13.491	**0.011**
Trait Anger	-0.159	-6.121 – 5.804	0.958	-1.466	-6.869 – 3.936	0.590	-0.449	-6.166 – 5.268	0.876	-1.294	-7.111 – 4.523	0.659
AUDIT	-1.274	-5.704 – 3.156	0.568	-1.525	-5.539 – 2.488	0.451	-1.574	-5.822 – 2.674	0.463	-0.511	-4.834 – 3.812	0.814
Ethnicity [Black [R], White]	-1.393	-11.337 – 8.550	0.783	4.379	-3.874 – 12.631	0.298	-3.607	-11.916 – 4.702	0.394	1.176	-6.959 – 9.312	0.776
Error Rate	39.286	-120.225 – 198.798	0.629	54.941	-76.785 – 186.667	0.413	38.202	-86.653 – 163.056	0.548	57.544	-87.899 – 202.988	0.437
Tendency [Approach [R], Avoid]	26.679	16.723 – 36.634	**<0.001**	19.737	11.476 – 27.998	**<0.001**	16.377	8.073 – 24.681	**<0.001**	13.161	5.043 – 21.278	**0.002**
Drink [Placebo [R], Alcohol]	-21.755	-37.493 – -6.016	**0.007**	-26.038	-42.815 – -9.262	**0.003**	-20.641	-35.166 – -6.116	**0.006**	-26.013	-41.371 – -10.656	**0.001**
Block 2: Interaction Term (Tendency * Drink)												
(Intercept)	683.219	540.610 – 825.827	**<0.001**	673.211	543.653 – 802.769	**<0.001**	670.134	533.499 – 806.770	**<0.001**	685.374	546.287 – 824.460	**<0.001**
Tendency [Approach [R], Avoid]	30.756	16.699 – 44.813	**<0.001**	24.390	12.726 – 36.054	**<0.001**	15.956	4.212 – 27.699	**0.008**	12.292	0.788 – 23.795	**0.036**
Drink [Placebo [R], Alcohol]	-17.705	-36.207 – 0.797	**0.061**	-21.354	-40.015 – -2.693	**0.025**	-21.066	-37.766 – -4.366	**0.014**	-26.883	-44.213 – -9.553	**0.003**
Tendency [Approach [R], Avoid] * Drink [Placebo [R], Alcohol]	-8.186	-28.104 – 11.732	0.420	-9.321	-25.822 – 7.179	0.268	0.845	-15.772 – 17.462	0.920	1.735	-14.525 – 17.995	0.834

Table continues on next page

	Anger		Happy		Sad		Disgust	
Random Effects								
	Variance	*SD*	*Variance*	*SD*	*Variance*	*SD*	*Variance*	*SD*
Residual	4146	64.39	2853	53.42	2893	53.79	2767	52.60
ID	10724	103.56	11036	105.05	9597	97.97	10522	102.58
Drink [Placebo=0, Alcohol=1]	2935	54.17	4292	65.52	2832	53.21	3436	58.62
ICC	0.70		0.76		0.75		0.77	
N	81_{ID}		81_{ID}		81_{ID}		81_{ID}	
Observations	648		648		648		648	
Marginal R^2 / Conditional R^2	0.091 / 0.724		0.092 / 0.780		0.079 / 0.772		0.086 / 0.791	

Note: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Ethnicity of facial expressions (Black, White), Error Rate is the proportion of mistakes made (i.e., responded with approach in avoidance cued trials and vice versa), Tendency (Approach, Avoid), Drink (Placebo, Alcohol). In Block 1, the fixed effects of tendency and drink were entered into a linear mixed effects model to test the main effects of tendency and drink on RT. In Block 2, a tendency by drink interaction term was entered into the model to test the interaction between tendency and drink on RT. Age, gender, trait anger AUDIT, error rate were also entered into each model to control for their influence. Random effects for each full model included random intercepts for subject ID and by ID random slopes for the effect of drink. P values for each fixed effect were estimated using Kenward-Rogers d.f.

[R] *Reference level for each categorical variable*

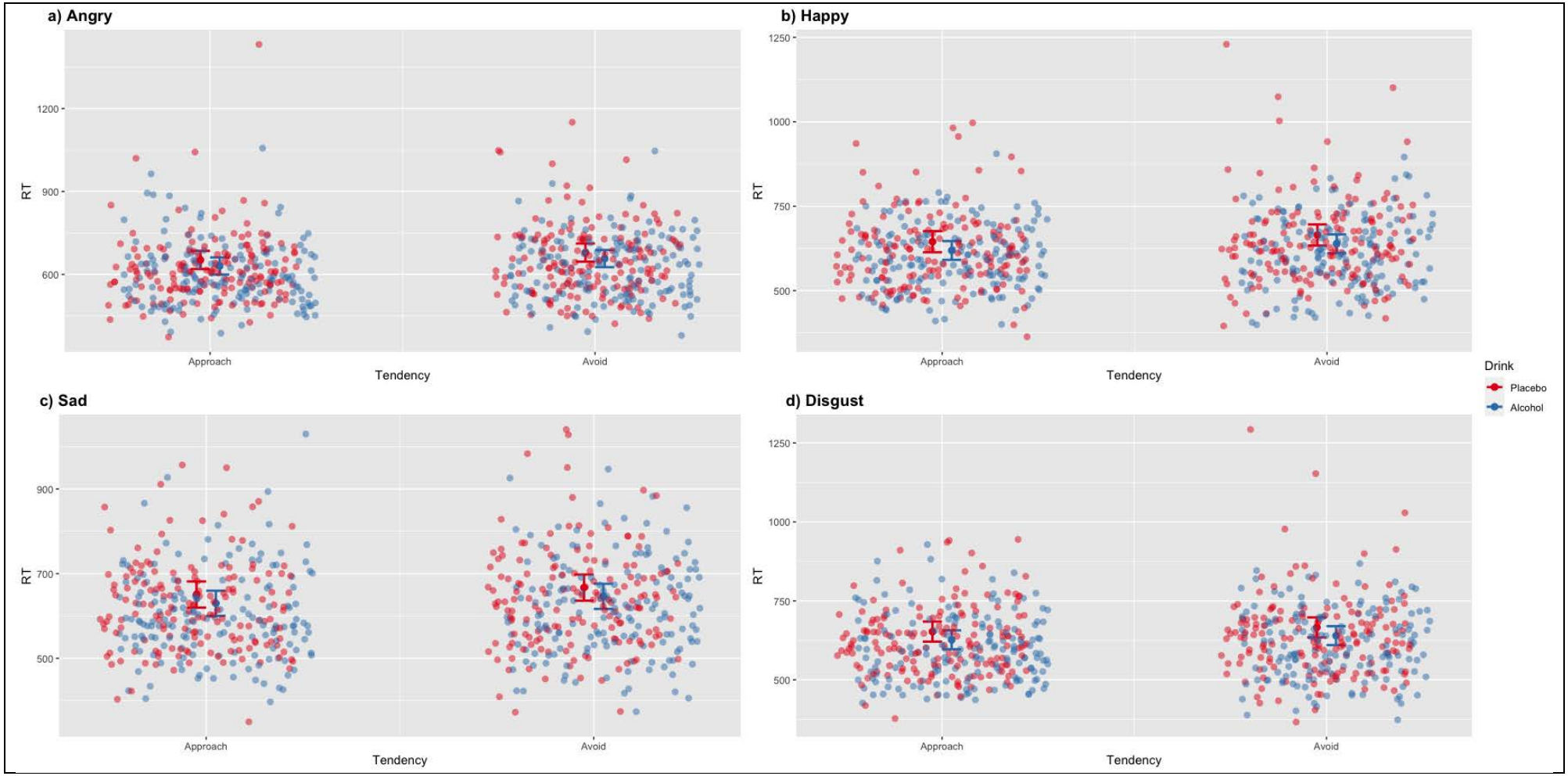

Figure 4.3: Reaction times (RT) when cued to approach and avoid emotional facial stimuli (Angry, Happy, Sad, Disgust) following alcohol and placebo drinks. Error bars represent 95% confidence intervals.

4.6. Discussion

The primary aim of this study was to investigate whether social drinkers demonstrate greater Hostile Attribution Bias towards emotional facial expressions following alcohol compared to placebo. In addition, the influence alcohol has on approach avoidance tendencies when seeing emotional faces was also investigated. For global hostility ratings, there was no evidence of an effect of alcohol suggesting that social drinkers do not see emotions as more hostile following acute alcohol compared to placebo. However, when considering emotional intensity, there was weak evidence to suggest that alcohol consumption influenced hostile attribution bias of emotional expression as the intensity of the emotion increased. This interaction specifically highlighted that low intensity emotions (i.e., emotionally ambiguous to the perceiver) were rated as more hostile following alcohol. As the intensity of the emotion increased, this alcohol induced difference reduced, resulting in the difference between alcohol and placebo hostility ratings diminishing as a function of intensity. Research suggests that ambiguous social information is often seen as more hostile (Milich & Dodge, 1984), and that alcohol consumption impairs global emotion processing (i.e., less recognition accuracy when intoxicated compared to sober) (Tucker & Vuchinich, 1983). It is likely that alcohol consumption leads to an increase in ambiguity of the emotion expressed by impairing the ability to accurately recognise the emotion, which results in greater hostile attribution bias. Seeing ambiguous facial expression as more hostile when intoxicated has social relevance as the propensity to see faces as more hostile may lead to increased aggression and violence (Wegrzyn et al., 2017). Similar past research indicates that higher levels of hostile attribution bias are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006) and consequently plays a role in reactive aggressive behaviour (Crick & Dodge, 1996). Therefore, if individuals see ambiguous facial expression as more hostile under the

influence of alcohol this may increase the likelihood of aggressive responding. These findings are also similar to those outlined in forensic populations (Schonenberg & Jusyte, 2014). Smeijers et al. (2017) similarly report a greater hostile attribution bias towards facial stimuli in typically aggressive individuals (i.e., forensic outpatient sample) compared to healthy controls. This suggests that the hostile judgements of ambiguous emotions following the acute consumption of alcohol may mimic these judgements made by aggressive individuals. Given the social context in which alcohol is often consumed, and the likelihood of alcohol increasing the ambiguity of facial expression recognition, hostile attribution bias may be one mechanism by which alcohol consumption leads to aggressive behaviour in otherwise normal social drinkers.

At an emotion specific level, happy facial expressions were seen as more hostile regardless of the emotion intensity displayed. As the happiness intensity increased, hostile judgements reduced however, ambiguous (i.e., low intensity) and non- ambiguous (i.e., high intensity) displays of happiness were similarly perceived as more hostile following alcohol compared to placebo. Happy faces being seen as more hostile when intoxicated is of social importance as it is considered to be a positive emotion (Calvo & Beltran, 2013). Therefore, the increased hostile perception of happy faces may increase the likelihood of aggression by reducing the perceivers exposure to positive cues that promote pro-social behaviour. The emotional intensity displayed in specific emotional faces was also important to consider as, previously mentioned, hostile attribution bias often manifests when observations of social context cues are seen to be ambiguous (Milich & Dodge, 1984). As emotional intensity of angry and disgusted faces increased, so did perceived hostility suggesting that the clearer the display of these emotions the more hostile they were seen. On the other hand, when the intensity of happy and sad emotions increased, hostility ratings reduced. These findings support the notion that hostility interpretation is conceptually different to

an anger perception bias (Deffenbacher, 2000; Eckhardt et al., 2004; Tsikandilakis et al., 2020) as the increased propensity to see hostility is not only limited to angry faces. There are a lot of facial characteristic similarities between an angry face and a disgusted one displayed in isolation (Wieser & Brosch, 2012), which may explain why both expressions were similarly rated as more hostile as intensity increased. When considering the drink by intensity interaction for these emotions, both angry and disgusted faces were rated as more hostile at low emotional intensities (i.e., ambiguous displays of anger and disgust) following alcohol compared to placebo. This drink related difference reduced as intensity increased (i.e., the increased hostility rating associated with alcohol diminished as the emotion displayed in the expressions became clearer). So, whilst ambiguous displays of anger and disgust were rated as more hostile following alcohol compared to placebo, high intensity displays of these emotions (i.e., unambiguous) were not. Wieser and Brosch (2012) highlight the importance of contextual cues when processing emotional facial expressions. This highlights a potentially interesting avenue for future research development. Giving the facial expression context may help to disentangle the hostility perception of angry and disgusted faces as without contextual cues they seem to be judged alike.

This study hypothesised that social drinkers would display greater tendency to approach a hostile stimulus (i.e., angry and disgusted faces) following alcohol compared to placebo. Findings indicate that this was not the case. There was evidence for a main effect of drink suggesting faster RTs following alcohol compared to placebo. There was also evidence for main effect of tendency suggesting a faster RT in approach compared to avoidance conditions for all emotional expression (angry, happy, sad, disgust), but this was not moderated by alcohol consumption (no evidence of an approach/avoidance tendency by drink interaction). This suggests that alcohol consumption generally makes people quicker on the AAT task (qualified by the main effects of drink on RTs),

and that individuals are faster to approach than avoid emotional stimuli (qualified by the main effects of tendency on RTs). But social drinkers show no increased/decreased tendency to approach or avoid an angry or disgusted face following alcohol compared to placebo. Similarly, for happy and sad faces, there was no evidence to suggest that the propensity to approach or avoid was moderated by acute alcohol consumption. These findings may be due to the nature of the approach/avoidance task used in the present study. Implicit instructions meant that participants were not required to attend to the emotional valence of the stimuli but were instead required to respond to a task-irrelevant feature (i.e., the border of the image displaying the emotional facial expression). Feedback was given on each trial in the form of image zooming. For approach trials this meant that images would increase in size and in avoidance trials they would decrease (Phaf et al., 2014). Effects established using this implicit version would suggest an automatic link between affective information processing (i.e., emotional valence) and approach/avoidance tendencies. As there was no evidence of a difference in the tendency to approach or avoid emotional stimuli (angry, happy, sad and disgusted faces), these findings suggest that behavioural motivation may not be an automatic response to these social cues. A review evaluating implicit vs explicit instructions in approach avoidance paradigms concludes that explicit instructions often yield bigger effects sizes (Krieglmeyer & Deutsch, 2010). In this version of the task, participants are required to explicitly evaluate the emotional valence of a stimuli by responding with an approach action (i.e., joystick pull) or avoidance action (i.e., joystick push) when seeing positive and negative stimuli, respectively. It may be that approach/avoidance tendencies under the influence of alcohol require the individual to make conscious evaluations of cues in order to judge whether the social information should be approached or avoided. Future research should investigate implicit vs

explicit instruction on approach/avoidance tendencies following alcohol to investigate whether processing of social stimuli is automatic or driven by the conscious evaluation of valence.

4.6.1. Conclusion

Our findings suggest that ambiguous emotional facial expressions are judged as more hostile following acute alcohol consumption compared to placebo. This global hostile attribution bias when seeing ambiguous faces may increase the likelihood of maladaptive behaviour, as the greater propensity to judge socially relevant stimuli as hostile has been shown to increase aggressive responding. At an emotion specific level, happy faces are seen as more hostile following alcohol. Happy is considered to be a positive emotion. If alcohol induces a hostile attribution bias of this emotion, positive social cues may be missed which may decrease pro-social behaviour and increase the likelihood of aggressive responding. This study failed to find an effect of alcohol and approach/avoidance tendencies when seeing emotional facial expression. More work is required to establish the behavioural motivations associated with hostile and non-hostile faces. This could involve investigating the precise context in which hostile displays of emotion become approached.

CHAPTER 5: EFFECTS OF ACUTE ALCOHOL CONSUMPTION ON HOSTILE ATTRIBUTION BIAS OF THIRD-PARTY ENCOUNTERS AND SOCIAL EVALUATION INFERENCE

Keywords: Dyadic social interaction; social evaluation; hostile attribution bias, acute alcohol consumption; alcohol related aggression.

5.1. Chapter Overview

This chapter aimed to investigate whether acute alcohol consumption effects hostile evaluations of dyadic social interactions (i.e., two people interacting) in social drinkers. Research suggests that alcohol influences hostile judgements of isolated facial expressions. A similar profile was expected when forming impressions of dyadic social interactions. This chapter also aimed to investigate how acute alcohol influences the way in which drinkers infer social-evaluative information about the self (self-referential) and others (other-referential) following feedback. It was anticipated that alcohol would increase perceived negative evaluations. Regular non-dependent drinkers (N=112) participated in a double-blind placebo-controlled experiment. Participants attended two sessions. In one, they consumed an alcoholic drink (0.4 g/kg), and in the other they consumed a matched placebo. In each session they completed two tasks. The first measured hostile ratings of dyadic social interactions (non-hostile, ambiguous, hostile). The second measured social-evaluative learning during and following a social interaction. Participants were required to evaluate whether a social agent liked them/others following three social rules (like, neutral, dislike). There was no evidence of a reduced hostile perception of dyadic social interactions following alcohol. Future research should explore specific attributes of a social interaction by manipulating whether individuals appear to know each other, and the perceived likeness between two individuals. Findings also suggest that social drinkers do not differ in how they process social evaluative information during a social interaction following alcohol and placebo drinks. They also suggest that alcohol does not influence the overall perceived likeness following the social interaction. Future research should also explore how other social evaluations are made based on similar cued feedback. Specifically, how threatening/hostile vs non-

threatening/non-hostile social feedback from a social agent influences evaluative inferences made following when intoxicated.

5.2. Introduction

Impressions are formed when observing others in a social interaction (Quadflieg & Penton-Voak, 2017). Most alcohol research to date focuses on impression formation when observing facial expressions of an isolated individual (Attwood & Munafo, 2014). The way individuals interact in a social situation, and the impressions formed by an observer are also likely to influence behaviour. Recent research to support this claim has shown that observing third party encounters of two people interacting within a social environment (i.e., dyadic social interactions) can directly influence the perceivers own behaviour and intentions towards others, even if the impressions formed are inaccurate (Quadflieg & Westmoreland, 2019). Therefore, dyadic interactions (two people interacting) have implications in relation to the success or failure of a social interaction. Individuals are often better at interpreting whether two people seen to be interacting in a dyad know each other (Latif et al., 2014; Place et al., 2009) but often struggle to judge the degree of rapport or how much they like each other (Bernieri & Gillis, 1995; Bernieri et al., 1996; Floyd & Erbert, 2003). This evidence may explain poor behavioural outcomes (i.e., aggression) following the observation of others. For example, the ability to distinguish between two people that know each other behaving aggressively (e.g., play fighting), and two people who do not know each other behaving aggressively (e.g., violent altercation) will differentially influence the perceivers behaviour and attitudes towards that interaction. Observations of third party encounters can also inform the perceiver of potential threat with regards to their own personal social standing (Mast & Hall, 2004), and this in turn can influence the tendency to approach or avoid a particular situation (Milinski, 2016). Observers can identify individuals prone to hostile, unsociable and potentially

dangerous behaviour (Hamlin, 2013). Therefore, perceived positive (e.g., caring, protection, co-operation) and negative (e.g., hostility, dangerous, unfair, volatile) interactions have the ability to influence the likelihood of the perceiver to interact with those who they observe (Quadflieg & Westmoreland, 2019).

Acute alcohol consumption has been shown to disrupt the processing of isolated emotional facial expressions (Attwood, Ohlson, et al., 2009; Craig et al., 2009) and impairs the overall accuracy of identifying the emotion displayed (Tucker & Vuchinich, 1983). What remains unclear is whether impressions/interpretations formed from viewing dyadic social interactions (i.e., observations of two or more individuals interacting) are similarly influenced by acute alcohol consumption. These interactions provide important social insights and alcohol-induced distribution to the processing of this information may increase the likelihood of maladaptive behaviour. In particular, hostile attribution bias theory suggests that alcohol related aggression may be due to the misinterpretation of ambiguous or innocuous social information as provocation or threat (Bartholow & Heinz, 2006). Therefore, ambiguous dyadic interactions may also be misinterpreted in a similar manner which could increase the likelihood of aggressive responding. This study tests whether acute alcohol increases hostile evaluations of dyadic social interactions.

Another potential important cognitive mechanism within a social interaction is the ability to infer whether you are liked or disliked. Social interactions are dynamic with behaviour contingent on feedback received from others. Understanding how individuals use this feedback to learn and determine how others evaluate them within a social interaction may contribute to understanding the success or failure of a social interaction (Button et al., 2012). Research from the anxiety literature suggests an association between highly anxious individuals and a greater tendency to infer negative evaluations of themselves (i.e., others dislike them more) (Button et al.,

2015). These authors examined instrumental learning as it might occur during a social interaction, where the individual infers how the other social agent (i.e., the perceiver) evaluates them using cued feedback. What remains unclear, is whether alcohol consumption effects how individuals infer social evaluation of themselves from others' through associative learning (i.e., cued feedback). A poor ability to learn from feedback received from others during a social interaction could influence the interaction, as alcohol may reduce the perceived positive evaluations of themselves and subsequently increase the perceived negative evaluations. In the context of alcohol-related aggression, this may increase the likelihood of aggressive responding.

5.3. Aims

This study aims to address two primary objectives. Firstly, whether acute alcohol consumption affects hostile evaluations of dyadic social interactions (i.e., two people interacting) in social drinkers. In addition, hostile perception of different types of dyadic social interaction (i.e., non-hostile, ambiguous, hostile) following alcohol consumption was also explored. It was hypothesised that dyadic social interactions will be seen as more hostile following acute alcohol consumption compared to placebo. This effect is anticipated to be more pronounced in ambiguous interactions. This study also aimed to investigate whether alcohol consumption effects how social drinkers infer social-evaluative information about the self (self-referential) and others (other-referential). Two outcome measures were used to assess learning during a social interaction (i.e., % of positive responses and errors to criterion), and one outcome measure was used to assess global likeness once the social interaction has taken place. The first two outcome measures were captured during the learning phase of the social interaction. This highlights how well individuals use cued feedback during the interaction to determine whether they/or others are liked by another person. The global likeness outcome measure was captured after the interaction and highlights whether the

individual reported being liked by another person. Determining whether social-evaluative inferences following alcohol are specific to the self (how evaluations are perceived about themselves) and not others (how evaluations are perceived towards others) was also explored. It was hypothesised that, following alcohol, social drinkers will be poorer at using cued feedback during the learning phase of a social interaction resulting in increased perceived negative evaluation. Similarly, it was hypothesised that overall perceived likeness following the social interaction will be lower following alcohol compared to placebo.

5.4. Methods

5.4.1. Participants

Social alcohol drinkers (n = 112; 48.2% male) were recruited from the University of Bristol (staff and students) as well as the general population by means of existing email lists, poster advertisement and word of mouth. The inclusion criteria included: good physical and psychiatric health (self-report), aged between 18-40 and speak English as first language or equivalent level of fluency. To avoid including participants with little/no drinking experience or undiagnosed alcohol dependence, only individuals that consumed between 5 and 35 alcoholic units per week were included. One UK unit equals one 25 ml single measure of spirit (ABV 40%), or a third of a pint of beer (ABV 5-6%) or half a standard (175 ml) glass of red wine (ABV 12%) (NHS, 2018). The exclusion criteria were any individuals that reported a strong familial history of alcoholism (in parents and/or siblings) or that reported a history of psychiatric disorder (including drug addiction), alcohol consumption within 24 hours prior to testing or if their breath alcohol concentration (BrAC) was above zero (tested on arrival), and if they weighed less than 50kg if female or 60kg if male. Participants gave signed informed consent prior to taking part in the study. Participants were reimbursed £15 on completion of the study or were awarded equivalent course credits. The study was approved by the University of Bristol's Faculty of Science Human Research Ethics Committee (reference: 26091994122). The study protocol was pre-registered on the Open Science Framework (doi: 10.17605/OSF.IO/FYWZH).

5.4.2. Design

A double-blind placebo-controlled experimental design with one within-subjects factor of drink (0.4 g/kg alcohol, placebo) was used. The primary dependent measure for Dyadic Hostile Attribution Bias Task (HABT-D) was hostility intensity rating (i.e., the degree of hostility

perceived in each dyadic social interaction). To explore hostile attribution bias when seeing specific types of dyadic social interactions following alcohol, an additional within subject factor of image hostility (hostile, Non-Hostile, ambiguous) was used. The primary dependent measures for the Social Evaluation Learning Task (SELT) were % of positive responses and errors to criterion (i.e., learning phase outcomes), as well overall rating of likeness (i.e., global rating) (see 5.4.4 *Measures*). To explore whether the social-evaluative inferences are specific to the self and not others an additional within subject factor of referential condition was used (evaluations directed towards the self, evaluations directed towards others).

5.4.3. Drink

Drinks were prepared by a research collaborator who was independent of data collection and drink administration was double-blind. Alcohol content was dependent on participant weight. An upper limit of 90 kg was set so that participants weighing more than 90 kg received the same drink as a 90 kg participant. The alcoholic drinks were mixed using one-part vodka (37.5% ABV) to three parts tonic water. The dose used was 0.4 grams of alcohol per kg of body weight (g/kg (Attwood, Ataya, et al., 2009; Craig et al., 2009). Placebo drinks were matched volume tonic water. In order to mask the taste of alcohol, drinks were chilled and flavoured with lime cordial (40 ml) prior to serving. The inside rim of the glass was sprayed twice with a vodka mist.

5.4.4. Materials

5.4.4.1. Dyadic Hostile Attribution Bias Task (HABT-D)

The stimuli chosen to be used in the HABT-D display dyadic social interactions (i.e., two-person interaction) and were selected from Google images. Only images that were free to use, edit and share were used. A pilot study was conducted to measure perceived hostility of the images. This presented 105 images to 25 participants. They were required to rate the perceived level of

hostility using a visual analogue scale ranging from Non-Hostile (0) to Hostile (100). Mean ratings for each image that fell in the lower tertile were categorised as 'Non-Hostile' interactions and those in the upper tertile were categorised as 'Hostile'. Remaining images between the lower and upper tertiles were categorised as 'Ambiguous' (see Figure 5.1). The final stimuli set used in the HABT-D include 35 'Hostile', 35 'Non-Hostile' and 35 'Ambiguous' interaction images (105 images in total; see Figure 5.2). On each trial of the main task, a single image was displayed for 10,000ms or until a response was made (after which the trial was over). Each image was presented once (105 trials in total). The outcome measure of interest was the % of hostile responses.

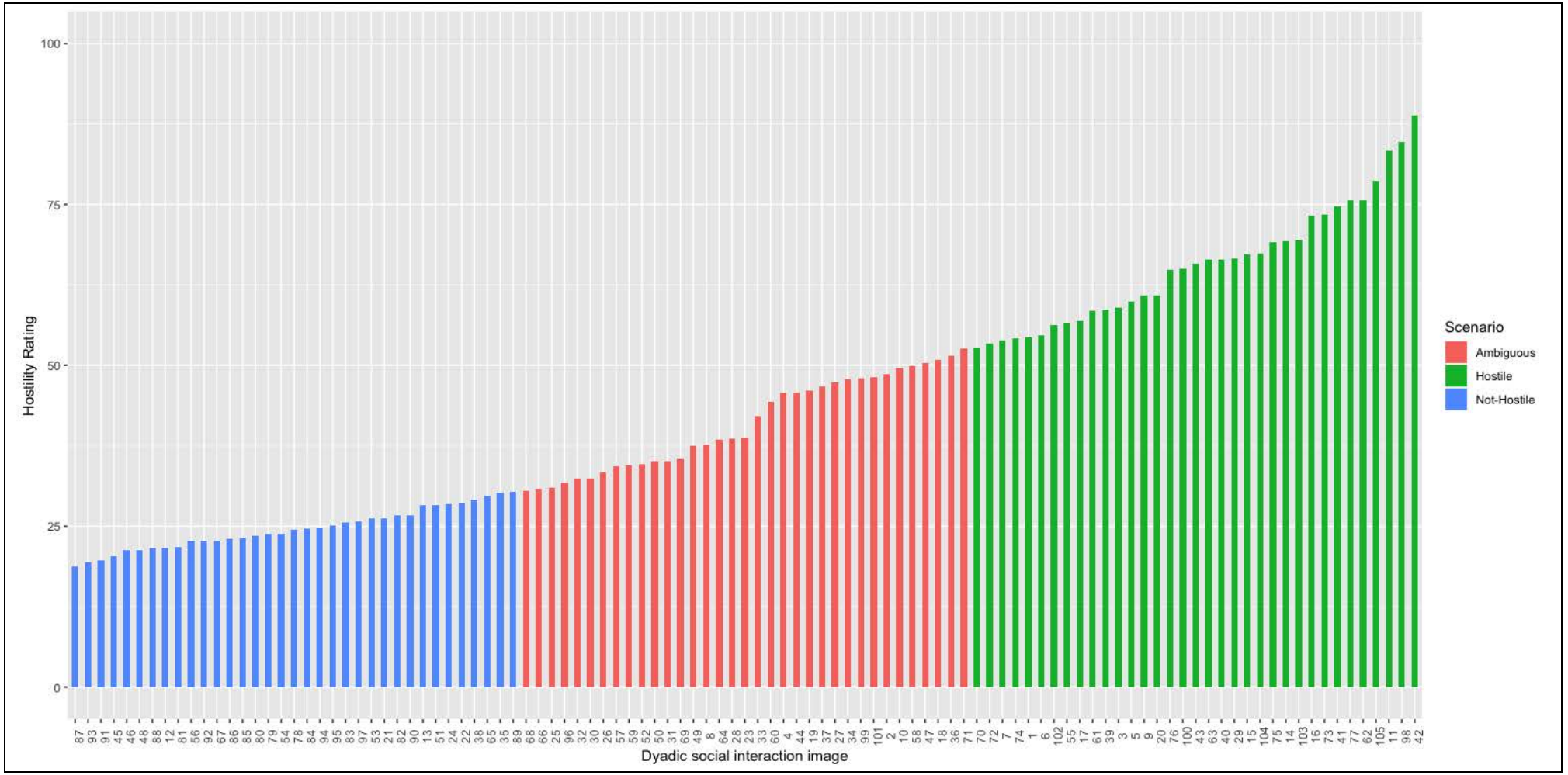

Figure 5.1: Mean hostility ratings of each dyadic social interaction image. Scenario categories defined using tertiles (the lower tertile images were categorised as 'Non-Hostile' interactions and those in the upper tertile were categorised as 'Hostile'. Remaining images between the lower and upper tertiles were categorised as 'Ambiguous'.

a)

b)

c)

Figure 5.2: Example images of the dyadic social interactions used in the HABT-D. a) Non-hostile interaction, b) ambiguous interaction and c) hostile interaction. Only images that were free to use, edit and share were selected.

5.4.4.2. Social Evaluation Learning Task (SELT)

This task was developed by Button et al. (2015) and was based on probabilistic stimulus-reward tasks (Button, Browning, Munafo, & Lewis, 2012; Chamberlain et al., 2006). It involves the participant meeting six computer personas, during six test blocks. Each of these personas requires the participants to learn one of three social rules (i.e., person is liked by persona, neutral or disliked by persona) in one of two referential conditions (i.e., persona likes me [participants], persona likes other [e.g., George]). Each test block consists of a learning phase and a global rating phase. The learning phase simulates a social interaction and the global phase measures overall learning. At the start of each block consisting of 32 trials, participants meet a new persona who presents a series of positive/negative word pairs (e.g., witty-dull); word pairs are selected at random without replacement from a list of 64 pairs. On each trial, the participant was required to select one of the words from each pair that corresponds with what the persona thought about either the participant (i.e., self-referential) or a third individual "George" (i.e., other-referential). Participants are provided with feedback as to whether their decision was correct and are instructed to use trial and error to determine whether the persona likes/dislikes them/ "George". The social rules are implemented as follows: like (positive word correct 80% of the time), neutral (positive word correct 50%) and dislike (positive word correct 20%). At the end of each block, the persona asked the participant to provide a global rating of likeness (i.e., how much the person likes them/ "George"), using a scale rating (0 completely dislike – 100 completely like). The outcome measures of interest were % of positive responses during the learning phase and the global rating. For the like and dislike rules, errors to criterion were also calculated, which were the number of rule-incongruent words chosen (i.e., errors) before reaching the criterion of selecting 8 consecutive rule-congruent words. Where the criterion was not met total errors were used.

5.4.4.3. Questionnaires

Questionnaire measures included the Alcohol Use Disorders Identification Test (AUDIT) (Saunders, Aasland, Babor, Delafuente, & Grant, 1993), State-Trait Anger Expression Inventory (STAXI-2) (Spielberger, 1999), Positive and Negative Affect Schedule (PANAS) (Watson, Clark, & Tellegen, 1988), Patient Health Questionnaire – 9 (PHQ-9) (Kroenke, Spitzer, & Williams, 2001), and the Brief Fear of Negative Evaluations – II (BFNE-II) (Carleton, Collimore, & Asmundson, 2007).

5.4.5. Procedures

Eligible participants were required to attend two testing sessions (approximately 60 minutes each), scheduled at least 7 days apart. In session one, participants were given the opportunity to read the information sheet again and ask questions, before providing written informed consent. The researcher conducted a short screening procedure to verify eligibility. A short screening procedure to verify eligibility was conducted, which included measures of weight and an alcohol breath test (Draeger AlcoDigital 3000 Breathalyzer) to confirm zero breath alcohol concentration (BrAC). Participants were given the opportunity to reschedule should their BrAC be above zero. Weight information was passed to a research collaborator for drink preparation. Participants began by completing a short series of questionnaires including the AUDIT, STAXI-2, PANAS, PHQ-9, BFNE-II, SES measures. During both testing sessions, participants were given a single drink to consume. In session one, this contained either 0.4 g/kg alcohol or a matched placebo. The opposite drink was administered in session two (order counterbalanced). Participants were given 10 minutes to consume all of their drink and a further 10 minutes to sit quietly to allow for absorption. Next, participants completed the HABT-D followed by the SELT (order fixed), in which both tasks took approximately 10 minutes each to complete. They then completed a second

series of questionnaires including S-Ang subscale of the STAXI-2, PANAS, BFNE-II measures, and a second BrAC reading (in session one, this was recorded by a trained research assistant to avoid unblinding). Before leaving the session, participants were required to read and sign a post-session safety form, were offered the opportunity to stay behind until they feel any effects of alcohol had worn off and were offered a taxi home. At the end of session two, participants were debriefed, unblinded and reimbursed.

5.4.6. Sample Size Calculation

A sample size calculation was made based on preliminary data (n=74) from our laboratory (currently unpublished) that explored HAB towards emotional facial expressions. This data indicates an emotion specific change in HAB with happy faces interpreted as more hostile following alcohol (M = .08, SD = .13) when compared to placebo (M = .06, SD = .11). This suggested an effect size of dz = .26 (correlation between conditions r = .66). Based on these data, we would need a total sample size of 125 participants to achieve 80% power (alpha level of 5%) to observe an effect of alcohol on HAB towards dyadic social interactions of a similar size. As we are stratifying by gender, we aimed to recruit 126 participants (50% male). Interaction analyses were exploratory.

5.4.7. Statistical Analysis

The target for data collection (i.e., N=126) was not achieved due to the interruption of data collection caused by the coronavirus pandemic. Data from 112 participants were used in these analyses. Statistical analyses were conducted using R Studio (2019) (r version: R Core Team). One participants' data was removed from the HABT-D analysis and four participants data was removed from the SELT analysis due to incomplete and missing task data. It was planned that the HABT-D data would be analysed using a 2 drink (alcohol, placebo) x 3 interaction type (Hostile, Ambiguous,

Non-Hostile) repeated measures ANOVA with interactions being explored using t-tests. However, it was later decided that this data would be analysed using linear mixed effects (LME) modelling (Baayen et al., 2008). This allows for the systematic control over the random between-subject's variance whilst controlling for other fixed effect variance (age, gender, trait anger, AUDIT). Multivariate normality, homoscedasticity and multicollinearity assumptions were satisfied unless otherwise stated. The primary objective of this research was to investigate whether alcohol influences Hostile Attribution Bias when seeing dyadic social interactions and this study was powered to detect an effect of drink (i.e., alcohol vs placebo). An LME model (*Model 1: Main Effects Model*) with the fixed effects of alcohol (alcohol, placebo) and dyadic scenario (non-hostile, ambiguous, hostile) was used to test the main effects of drink and dyadic scenario (lme4 package in r: Bates et al., 2015). As the dyadic scenario main effect has three categorical levels, dummy coding was used to contrast each level (Ambiguous, Hostile) with a reference category (Non-Hostile). Age, gender, trait anger and AUDIT scores were also entered as fixed effects to adjust for their influence. Random intercepts were entered for subject ID as random effects. P values for each fixed effect were estimated using Kenward-Roger degrees of freedom (Luke, 2017). To explore the interaction between drink and dyadic social interaction a second LME model was used (*Model 2: Interaction Model*). A drink by image scenario interaction term was entered as a fixed factor. Age, gender, trait anger and AUDIT scores were again entered as fixed effects to adjust for their influence. Random intercepts for subject ID, as well as by ID random slopes for the effect of drink were entered as random effects. P values for the interaction term were estimated using Kenward-Roger degrees of freedom (Luke, 2017). Interactions were explored using planned contrasts where categorical interactions were compared.

It was planned that the SELT data would be analysed using a 2 drink (alcohol, placebo) x 3 rule (like, neutral, dislike) x 2 referential condition (evaluations directed towards the self, evaluations directed towards others) to investigate % positive responses and global likeness rating. And for errors to criterion, a 2 drink (alcohol, placebo) x 2 rule (positive, negative) x 2 referential condition (evaluations directed towards the self, evaluations directed towards others) would be used (neutral rule omitted). All interactions would be explored using t-tests. Similarly, it was later decided that this data would be analysed using linear mixed effects (LME) modelling (Baayen et al., 2008). To visualise the learning process, the cumulative mean positive responses for the 32 trials for the alcohol and the placebo conditions following each rule (dislike, neutral, like) were plotted (self and other referential conditions plotted separately, *see* Figure 5.4*)*. An LME model (*Model 1: Main Effects Model*) with the fixed effects of alcohol (alcohol, placebo), rule (like, neutral, dislike) and referential condition (self, other) was used to test the main effects of drink, rule and referential condition on % of positive response and global rating outcome measures. As the rule main effect has three categorical levels, dummy coding was used to contrast each level (Neutral, Like) with a reference category (Dislike). Age, gender, trait anger and AUDIT scores were also entered as fixed effects to adjust for their influence. To explore the interaction between drink, rule and referential condition a second LME model was used (*Model 2: Interaction Model*); a drink by rule by referential condition interaction term was entered as a fixed effect. Age, gender, trait anger and AUDIT scores were again entered as fixed effects to adjust for their influence. The same analysis strategy was used for errors to criterion with the exception of the neutral rule being omitted (i.e., only like and dislike rules were entered into each model for the rule condition). For % of positive responses, errors to criterion and global ratings, both models 1 and 2 had random intercepts for subject ID entered as a random effects. P values for main effects and the interaction

terms were estimated using Kenward-Roger degrees of freedom (Luke, 2017). Three-way and two-way interactions were explored using planned contrasts.

5.5. Results

5.5.1. Participant Characteristics

Participant were aged between 18-33 (M= 20.2, SD= 2.3) and weighed between 50-105kg (M= 69.8 SD= 12.0). Scores on the AUDIT ranged from 2-34 (M= 12.3, SD= 5.9) and scores on the trait aggressive subscale of the STAXI-2 ranged from 10-28 (M= 15.3, SD= 3.4). When asked on completion of each testing session, 90.2% of participants believed they had consumed alcohol when the drink administered was alcohol. In comparisons, 25.2% believed they had consumed alcohol when the drink administered was placebo.

5.5.2. Hostile Attribution Bias of Dyadic Social Interactions

Table 5.1 displays descriptive statistics for the interaction between drink and dyadic social interaction scenario. Model 1 was used to test the main effects of drink and dyadic scenario on hostile ratings of dyadic social interactions; Table 5.2 displays the model estimates. There was no evidence for a main effect of drink on hostile ratings of dyadic social interactions ($p = .400$). There was strong evidence for a main effect of image scenario on hostile ratings of dyadic social interactions ($p < .001$). Estimates suggest that ambiguous and hostile dyadic interaction were seen as 24.0% and 50.3% more hostile (respectively), when compared to non-hostile interactions ($ps < .001$). Model 2 was used to test the interaction between drink and dyadic scenario on hostile ratings of dyadic social interactions; Table 5.2 displays the model estimates. There was no evidence of a drink by image scenario interaction ($p = .568$) suggesting that alcohol consumption does not differentially influence hostility ratings of non-hostile, ambiguous and hostile dyadic social interactions (see Figure 5.3). LME model 1 and 2 both adjusted for the influence of age, gender, trait aggression, AUDIT score. There was modest evidence for an effect of trait anger on hostile attribution bias ($p = .017$); estimates suggest a 0.7% increase in hostility rating for every unit

increase in trait anger score. There was weak evidence for an effect of gender suggesting males rated dyadic social interactions as 3.6% less hostile compared to females (*p =.085*). There was no evidence for an effect of age or AUDIT score (*ps >.296*).

Table 5.1: Mean and standard deviation (SD) for Hostility ratings of dyadic social interactions (Non-Hostile, Ambiguous, Hostile) following alcohol and placebo drinks.

Image	**Alcohol**		**Placebo**	
	Mean	**SD**	**Mean**	**SD**
Non-Hostile	10.85	16.74	9.86	14.56
Ambiguous	34.82	26.35	33.93	25.50
Hostile	60.37	28.12	60.79	26.84
Average	35.35	23.74	34.86	22.30

Notes: n=111

Table 5.2: LME model estimates, 95 % Confidence intervals, and p-values for global hostile ratings of dyadic social interactions. Random effect variance for subject ID.

Fixed Effects

	Model 1: Main Effects Model (Drink + Dyadic Scenario)			**Model 2: Interaction Model (Drink * Dyadic Scenario)**		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
(Intercept)	-1.172	-19.576 – 17.232	0.900	-1.448	-19.872 – 16.976	0.876
Age	0.247	-0.529 – 1.023	0.529	0.247	-0.529 – 1.023	0.529
Gender [Female [R], Male]	-3.593	-7.690 – 0.504	**0.085**	-3.593	-7.690 – 0.504	**0.085**
Trait Anger	0.673	0.125 – 1.221	**0.017**	0.673	0.125 – 1.221	**0.017**
AUDIT	-0.189	-0.546 – 0.168	0.296	-0.189	-0.546 – 0.168	0.296
Drink [Placebo [R], Alcohol]	0.528	-0.705 – 1.761	0.400	1.080	-1.057 – 3.217	0.321
Dyadic Scenario [Non-Hostile [R], Ambiguous, Hostile]			**<0.001**			**<0.001**
Dyadic Scenario [Ambiguous]	23.986	22.476 – 25.496	**<0.001**	24.067	21.930 – 26.205	**<0.001**
Dyadic Scenario [Hostile]	50.261	48.751 – 51.771	**<0.001**	51.007	48.869 – 53.144	**<0.001**
Dyadic Scenario [Non-Hostile [R], Ambiguous, Hostile] * Drink [Placebo [R], Alcohol]						0.568
Dyadic Scenario [Ambiguous] * Drink [Alcohol]				-0.163	-3.186 – 2.859	0.915
Dyadic Scenario [Hostile] * Drink [Alcohol]				-1.492	-4.515 – 1.531	0.333

Table continues on next page

	Model 1: Main Effects Model (Drink + Dyadic Scenario)		Model 2: Interaction Model (Drink * Dyadic Scenario)	
Random Effects				
	Variance	*SD*	*Variance*	*SD*
Residual	65.61	8.100	76.99	8.775
ID	77.01	8.776	65.71	8.106
ICC	0.54		0.54	
N	111 $_{ID}$		111 $_{ID}$	
Observations	666		666	
Marginal R^2 / Conditional R^2	0.751 / 0.886		0.751 / 0.885	

Notes: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Drink (Placebo, Alcohol), Dyadic scenario (Non-Hostile, Ambiguous, Hostile). In model 1, the fixed effects of drink and dyadic scenario were entered into a linear mixed effects model. In model 2, the drink by dyadic scenario interaction term was entered into a linear mixed effects model. Age, gender, trait anger and AUDIT were also entered into both models as fixed effects to control for their influence. Random effects included random intercepts for subject ID. P values for each fixed effect were estimated using Kenward-Roger d.f.

^{R} *Reference level for each categorical variable.*

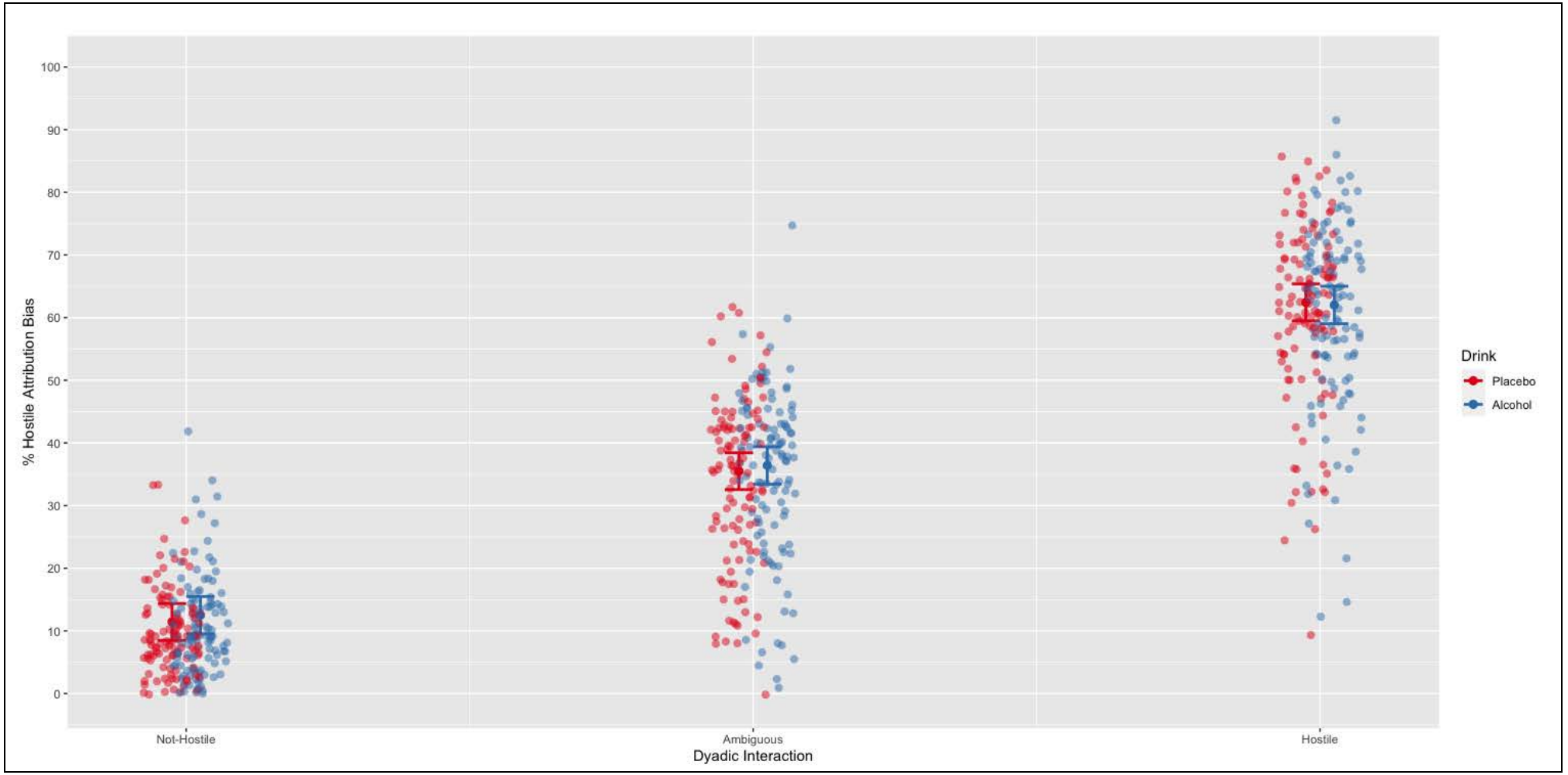

Figure 5.3: % Hostile responses when seeing not-hostile, ambiguous and hostile dyadic interaction scenarios following alcohol and placebo drinks. Error bars represent 95% confidence intervals.

5.5.3. Social Evaluation Learning

5.5.3.1. Learning Phase

5.5.3.1.1. % of Positive Responses

Figure 5.4 shows the cumulative mean % positive responses over the 32 learning trials for each of the rules (dislike, neutral, like) and referential condition (self, other) following alcohol and placebo drinks. These curves show that participants did adjust their behaviour on a trial-by-trial basis to learn each of the rules. As expected, responses varied over the initial few trials. After these initial trials, the alcohol curve in the dislike rule condition shows an increase in positive responses compared to placebo for self-referential learning. A similar trend can be seen following alcohol in the like rule condition compared to placebo for other referential learning. Alcohol also appears to reduce positive responses in the neutral rule condition compared to placebo for the self-referential learning. Participants behave similarly in all rule conditions for both self and other referential learning. Table 5.3 displays descriptive statistics for the interaction between drink, rule and referential condition for % of positive responses (i.e., learning phase). Model 1 was used to test the main effects of drink (alcohol, placebo), rule (dislike, neutral, like) and referential condition (self, other); Table 5.4 displays the model estimates. There was no evidence for a main effect of drink on % of positive responses ($p = .882$). There was strong evidence for a main effect of rule ($p < .001$). Contrasts show that the % of positive responses in the neutral and like conditions were an estimated 42.4% and 47.1% higher (respectively) than the dislike condition ($ps < .001$). There was evidence for a main effect of referential condition showing a 3.0% increase in % of positive responses in the self compared to other referential condition. Model 2 was used to test the two-way and three-way interactions between drink, rule and referential condition on % of positive responses; estimates are displayed in Table 5.4. There was no evidence for a two-way interaction

between drink and rule (*p =.344*, see Figure 5.5). There was modest evidence for a two-way interaction between rule and referential condition (*p =.012*, see Figure 5.6). Contrasts show that following the neutral rule % of positive responses increased by 7.5% in the self compared to the other referential condition (*ps =.018*). There was no evidence of a difference following the dislike and like rules for other and self-referential conditions (*p =.282*). There was no evidence of a two-way interaction between drink and referential condition (*p =.966*) or three-way interaction between drink, rule and referential condition interaction (*p= .863*) on % of positive responses.

Table 5.3: Mean (M) and standard deviation (SD) for % of positive responses, errors to criterion and global ratings following alcohol and placebo drinks in each rule conditions (dislike, neutral, like) for self and other referential learning.

Outcome Measure	**Referential Condition**	**Rule**	Alcohol		Placebo	
			M	SD	M	SD
% Positive response rate	Self	Like (80%)	78.70	15.40	78.40	15.50
		Neutral (50%)	75.00	17.30	77.00	15.70
		Dislike (20%)	31.60	21.10	29.70	20.80
	Other	Like (80%)	76.80	17.70	75.50	16.30
		Neutral (50%)	68.80	16.60	70.20	16.20
		Dislike (20%)	30.90	18.80	30.30	18.30
Error to Criterion	Self	Like (80%)	5.40	5.69	4.99	5.64
		Dislike (20%)	8.48	7.31	7.56	7.24
	Other	Like (80%)	5.19	6.44	5.94	6.05
		Dislike (20%)	8.19	6.61	8.01	6.49
Global rating	Self	Like (80%)	68.44	18.22	68.35	17.61
		Neutral (50%)	59.82	19.20	64.68	16.13
		Dislike (20%)	20.09	13.78	20.46	12.35
	Other	Like (80%)	66.70	20.42	66.79	18.85
		Neutral (50%)	60.18	17.74	61.10	19.02
		Dislike (20%)	22.94	11.97	23.12	11.52

Notes: n=108. For error to criterion, the neutral rule was omitted.

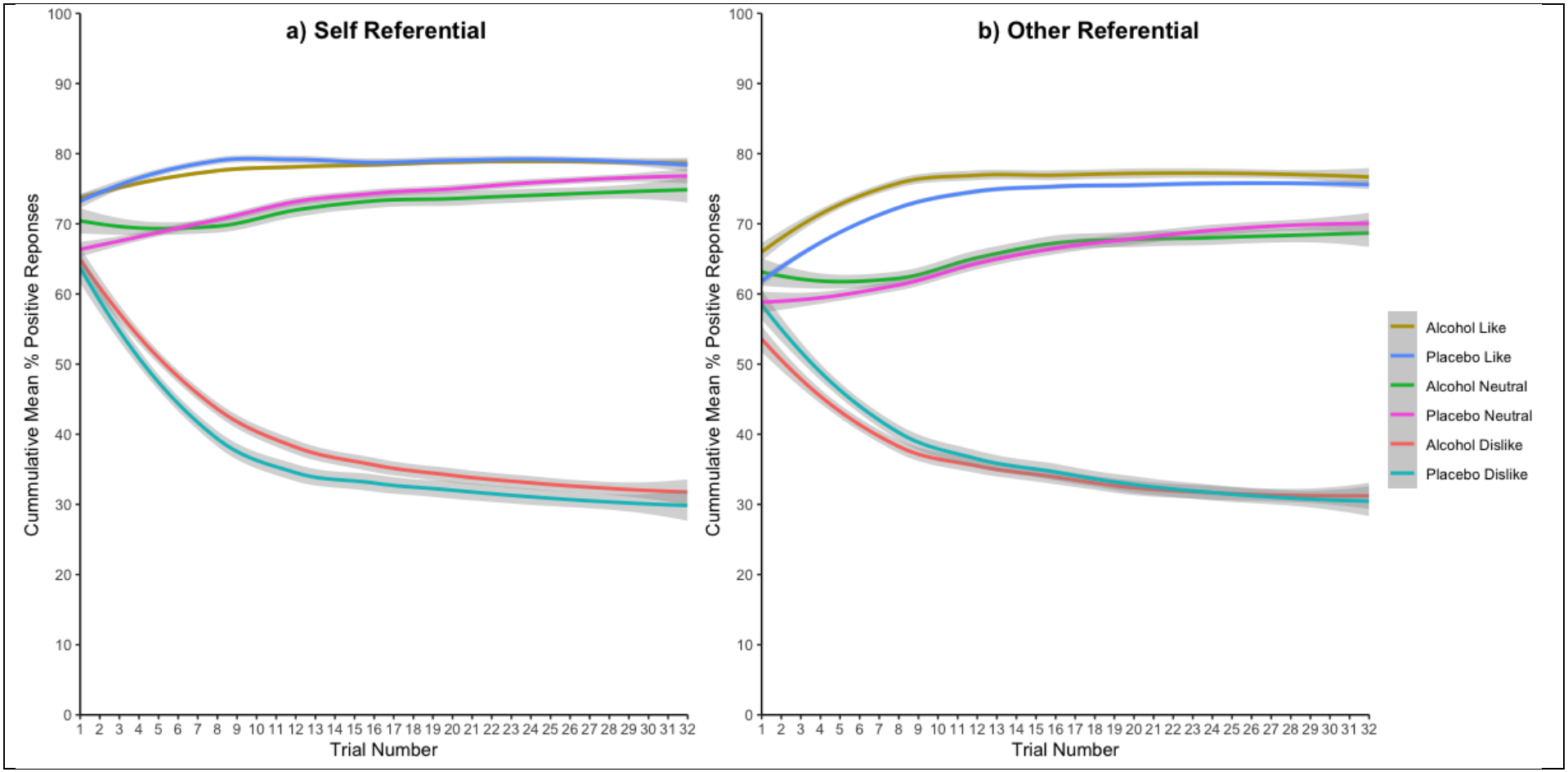

Figure 5.4: Learning curves following both alcohol and placebo drinks in each rule condition (Dislike, Neutral, Like) for a) self-referential learning and b) other referential learning conditions. The differentiation of the curves by rule condition show that participants were adjusting their responses based on feedback received, clearly demonstrating learning of each rule.

Table 5.4: LME model estimates, 95 % Confidence intervals, and p-values for % of positive responses (i.e., learning phase). Random effect variance for subject ID.

Fixed Effects

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			**Model 2: Interaction Model (Drink * Referential Condition * Rule)**		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
(Intercept)	28.658	14.125 – 43.190	**<0.001**	26.292	11.576 – 41.009	**0.001**
Age	0.153	-0.462 – 0.768	0.622	0.153	-0.462 – 0.768	0.622
Gender [Female [R], Male]	-1.984	-5.210 – 1.242	0.225	-1.984	-5.210 – 1.242	0.225
Trait Anger	0.122	-0.304 – 0.548	0.570	0.122	-0.304 – 0.548	0.570
AUDIT	-0.091	-0.371 – 0.189	0.521	-0.091	-0.371 – 0.189	0.521
Drink [Placebo [R], Alcohol]	0.135	-1.648 – 1.918	0.882	1.939	-2.421 – 6.298	0.383
Referential Condition [Self [R], Other]	-3.000	-4.782 – -1.217	**0.001**	0.579	-3.781 – 4.938	0.795
Rule [Dislike [R], Neutral, Like]			**<0.001**			**<0.001**
Rule [Neutral]	42.390	40.206 – 44.574	**<0.001**	47.627	43.268 – 51.987	**<0.001**
Rule [Like]	47.070	44.887 – 49.254	**<0.001**	48.987	44.628 – 53.347	**<0.001**
Rule [Dislike [R], Neutral, Like] * Drink [Placebo [R], Alcohol]						0.344
Rule [Neutral] * Drink [Alcohol]				-3.964	-10.129 – 2.201	0.207
Rule [Like] * Drink [Alcohol]				-1.562	-7.727 – 4.602	0.619
Rule [Dislike [R], Neutral, Like] * Referential Condition [Self [R], Other]						**0.012**
Rule [Neutral] * Referential Condition [Other]				-7.465	-13.630 – -1.300	**0.018**

Table continues on next page.

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			Model 2: Interaction Model (Drink * Referential Condition * Rule)		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
Rule [Like] * Referential Condition [Other]				-3.385	-9.550 – 2.780	0.282
Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.966
Drink [Alcohol] * Referential Condition [Other]				-1.302	-7.467 – 4.863	0.679
Rule [Dislike [R], Neutral, Like] * Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.863
Rule [Neutral] * Drink [Alcohol] * Referential Condition [Other]				1.910	-6.809 – 10.628	0.667
Rule [Like] * Drink [Alcohol] * Referential Condition [Other]				2.228	-6.491 – 10.947	0.616

Random Effects

	Variance	*SD*	*Variance*	*SD*
Residual	267.55	16.357	266.59	16.33
ID	30.73	5.543	30.81	5.55
ICC	0.10		0.10	
N	108 $_{ID}$		108 $_{ID}$	
Observations	1296		1296	
Marginal R^2 / Conditional R^2	0.603 / 0.644		0.605 / 0.646	

Notes: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Drink (Placebo, Drink), Referential Condition (Self, Other), Rule (Dislike, Neutral, Like). In model 1, the fixed effects of drink, referential condition and rule were entered into a linear mixed effects model. In model 2, a drink by referential condition by interaction term was entered into a linear mixed effects model. Age, gender, trait anger and AUDIT were also entered into both models as fixed effects to control for their influence. Random effects included random intercepts for subject ID. P values for each fixed effect were estimated using Kenward-Roger d.f.
[R] Reference level for each categorical variable.

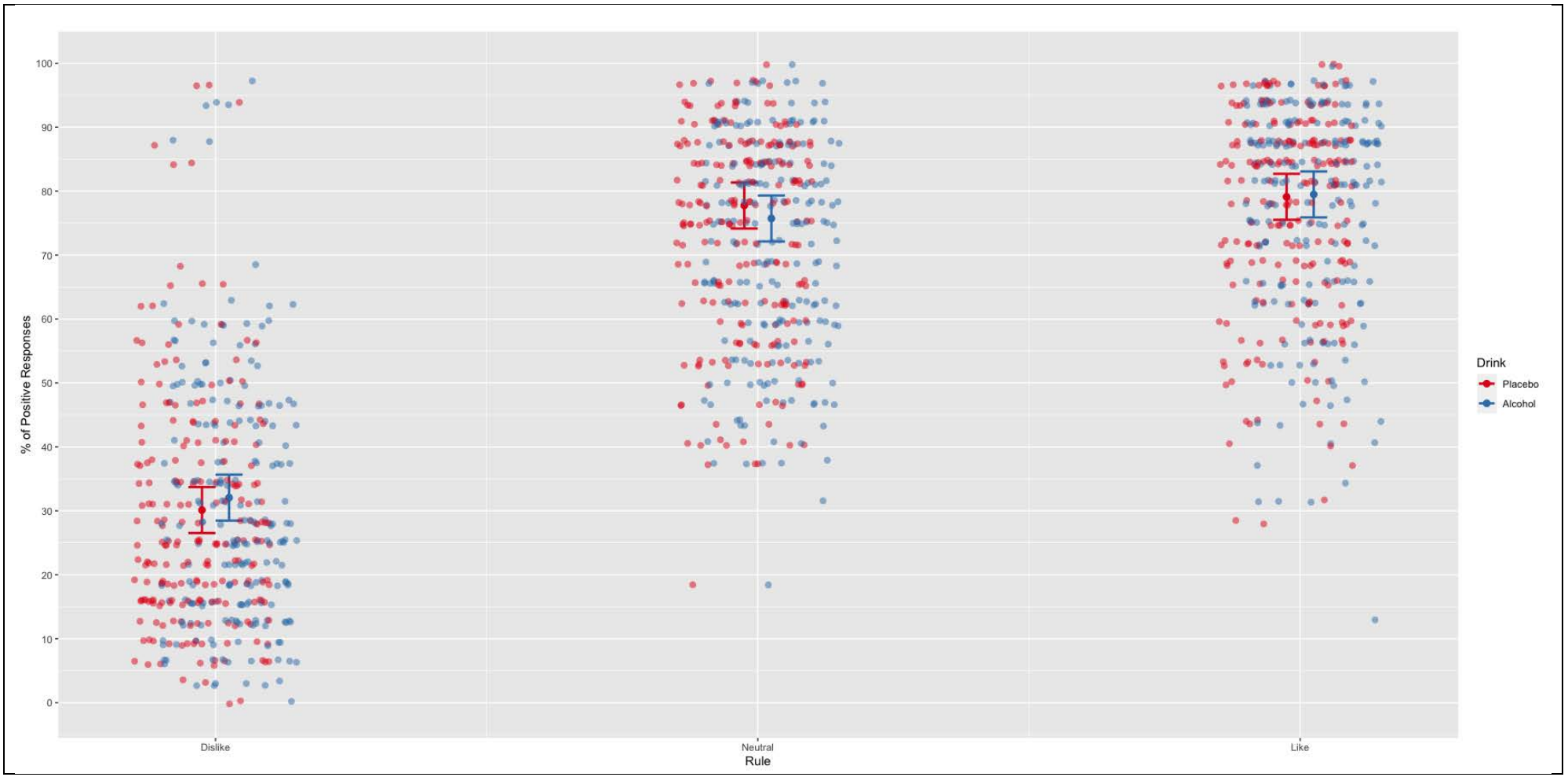

Figure 5.5: % of positive responses in the dislike, neutral and like rule conditions following both alcohol and placebo drinks. Error bars represent 95% confidence intervals.

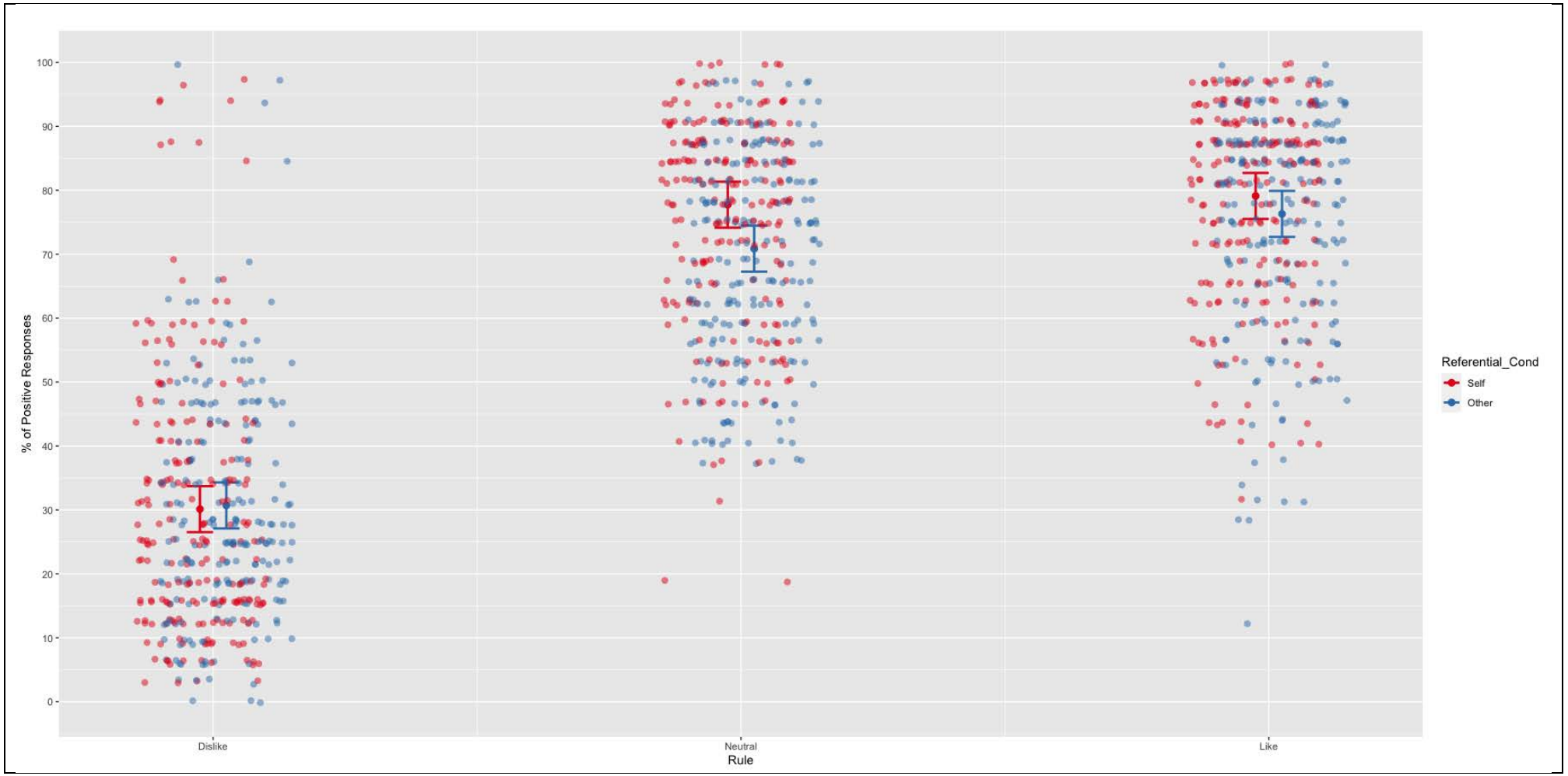

Figure 5.6: % of positive responses in the dislike, neutral and like rule conditions for self and other referential learning. Error bars represent 95% confidence intervals.

5.5.3.1.2. Errors to criterion

Table 5.5Table 5.3 displays descriptive statistics for the interaction between drink, rule and referential condition for errors to criterion. Model 1 was used to test the main effects of drink (alcohol, placebo), rule (dislike, like) and referential condition (self, other); Table 5.5 displays the model estimates. There was no evidence for a main effect of drink on errors to criterion (p =0.638). There was strong evidence for a main effect of rule (p <.001). Contrasts show that the individuals made an estimated 2.7 fewer errors before reaching the criterion (selecting 8 consecutive rule-congruent words) in the like compared to the dislike condition (*ps* <.001) demonstrating faster learning. There was no evidence for a main effect of referential condition (p =0.573). Model 2 was used to test the two-way and three-way interactions between drink, rule and referential condition on errors to criterion; estimates are displayed in Table 5.5. There was no evidence of any two-way (*ps* >.234) or three-way interaction (p= .785) on errors to criterion (see Figure 5.7).

Table 5.5: LME model estimates, 95 % Confidence intervals, and p-values for error to criterion (i.e., learning phase). Random effect variance for subject ID.

Fixed Effects

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			**Model 2: Interaction Model (Drink * Referential Condition * Rule)**		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
(Intercept)	8.096	1.261 – 14.930	**0.021**	7.804	0.926 – 14.682	**0.027**
Age	-0.084	-0.374 – 0.206	0.568	-0.084	-0.374 – 0.206	0.568
Gender [Female [R], Male]	0.227	-1.294 – 1.748	0.768	0.227	-1.294 – 1.748	0.768
Trait Anger	0.024	-0.177 – 0.225	0.814	0.024	-0.177 – 0.225	0.814
AUDIT	0.079	-0.053 – 0.211	0.235	0.079	-0.053 – 0.211	0.235
Drink [Placebo [R], Alcohol]	0.187	-0.594 – 0.969	0.638	0.917	-0.649 – 2.482	0.251
Referential Condition [Self [R], Other]	0.225	-0.557 – 1.006	0.573	0.444	-1.121 – 2.010	0.577
Rule [Dislike [R], Like]	-2.683	-3.465 – -1.901	**<0.001**	-2.574	-4.139 – -1.009	**0.001**
Rule [Dislike [R], Like] * Drink [Placebo [R], Alcohol]						0.362
Rule [Like] * Drink [Alcohol]				-0.509	-2.723 – 1.704	0.652
Rule [Dislike [R], Like] * Referential Condition [Self [R], Other]						0.715
Rule [Like] * Referential Condition [Other]				0.509	-1.704 – 2.723	0.652
Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.234
Drink [Alcohol] * Referential Condition [Other]				-0.731	-2.945 – 1.482	0.517
Rule [Dislike [R], Like] * Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.785

Table continues on next page.

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			Model 2: Interaction Model (Drink * Referential Condition * Rule)		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
Rule [Dislike [R], Like] * Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.785
Rule [Like] * Drink [Alcohol] * Referential Condition [Other]				-0.435	-3.566 – 2.695	0.785
Random Effects						
	Variance	*SD*		*Variance*	*SD*	
Residual	34.257	5.853		34.327	5.859	
ID	7.507	2.740		7.498	2.738	
ICC	264.78			16.272		
N	14.68			3.831		
Observations	264.78			16.272		
Marginal R^2 / Conditional R^2	14.68			3.831		

Notes: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Drink (Placebo, Drink), Referential Condition (Self, Other), Rule (Dislike, Neutral, Like). In model 1, the fixed effects of drink, referential condition and rule were entered into a linear mixed effects model. In model 2, a drink by referential condition by interaction term was entered into a linear mixed effects model. Age, gender, trait anger and AUDIT were also entered into both models as fixed effects to control for their influence. Random effects included random intercepts for subject ID. P values for each fixed effect were estimated using Kenward-Roger d.f.
[R]Reference level for each categorical variable.

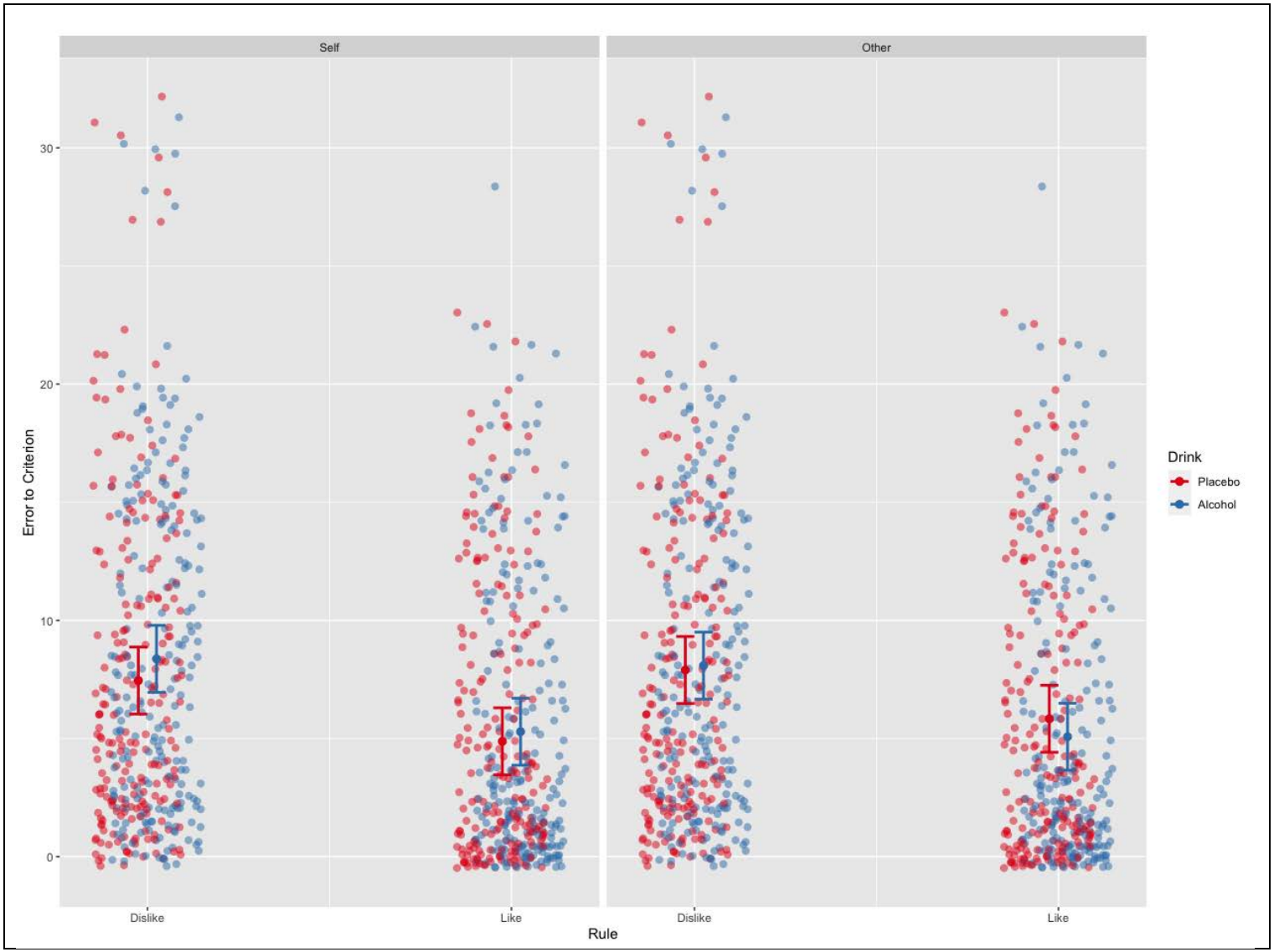

Figure 5.7: Error to criterion in self and other self-referential conditions for both the dislike and like rule conditions following both alcohol and placebo drinks. Error bars represent 95% confidence intervals.

5.5.3.2. Overall Learning (Global Rating)

Table 5.3 displays descriptive statistics for the interaction between drink, rule and referential condition for global ratings (i.e., overall learning). Model 1 was used to test the main effects of drink (alcohol, placebo), rule (dislike, neutral, like) and referential condition (self, other) on global ratings (i.e., overall learning); Table 5.6 displays the model estimates. There was no evidence for a main effect of drink or referential condition on global ratings (*ps >.246*). There was strong evidence for a main effect of rule (*p <.001*). Contrasts show that global ratings in the neutral and like conditions were an estimated 36.6% and 45.9% higher (respectively) than the dislike condition (*ps <.001*). Model 2 was used to test the two-way and three-way interactions between drink, rule and referential condition on global ratings; estimates displayed in Table 5.6. There was weak evidence of a two-way interaction between referential condition and rule (*p =.068*). Contrasts show that following the neutral rule, global likeness ratings in the self-referential learning condition was rated 6.3% higher than other-referential learning (*p= .044*); see Figure 5.8. There was no evidence of a global rating difference following the like and dislike rules when comparing self and other referential learning conditions (*ps >.165*). There was no evidence of a two-way interaction between drink and rule, or drink and referential condition (*ps >.339*). There was also no evidence of a three-way interaction between drink, rule and referential condition (*p =.950*).

Table 5.6: LME model estimates, 95 % Confidence intervals, and p-values for global ratings (i.e., overall learning). Random effect variance for subject ID.

Fixed Effects

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			**Model 2: Interaction Model (Drink * Referential Condition * Rule)**		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
(Intercept)	27.028	14.894 – 39.163	**<0.001**	25.207	12.854 – 37.561	**<0.001**
Age	-0.125	-0.636 – 0.387	0.631	-0.125	-0.636 – 0.387	0.631
Gender [Female [R], Male]	0.320	-2.366 – 3.006	0.814	0.320	-2.366 – 3.006	0.814
Trait Anger	-0.212	-0.566 – 0.143	0.239	-0.212	-0.566 – 0.143	0.239
AUDIT	0.067	-0.166 – 0.300	0.570	0.067	-0.166 – 0.300	0.570
Drink [Placebo [R], Alcohol]	-1.049	-2.823 – 0.724	0.246	-0.370	-4.711 – 3.970	0.867
Referential Condition [Self [R], Other]	-0.278	-2.051 – 1.496	0.759	2.593	-1.748 – 6.933	0.241
Rule [Dislike [R], Neutral, Like]			**<0.001**			**<0.001**
Rule [Neutral]	39.606	37.434 – 41.779	**<0.001**	44.074	39.734 – 48.415	**<0.001**
Rule [Like]	45.856	43.684 – 48.029	**<0.001**	47.870	43.530 – 52.211	**<0.001**
Rule [Dislike [R], Neutral, Like] * Drink [Placebo [R], Alcohol]						0.339
Rule [Neutral] * Drink [Alcohol]				-4.537	-10.675 – 1.601	0.147
Rule [Like] * Drink [Alcohol]				0.463	-5.675 – 6.601	0.882
Rule [Dislike [R], Neutral, Like] * Referential Condition [Self [R], Other]						**0.068**
Rule [Neutral] * Referential Condition [Other]				-6.296	-12.435 – -0.158	**0.044**

Table continues on next page.

	Model 1: Main Effects Model (Drink + Referential Condition + Rule)			Model 2: Interaction Model (Drink * Referential Condition * Rule)		
Predictors	*Estimates*	*95% CI*	*p*	*Estimates*	*95% CI*	*p*
Rule [Like] * Referential Condition [Other]				-4.352	-10.490 – 1.787	0.165
Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.452
Drink [Alcohol] * Referential Condition [Other]				0.185	-5.953 – 6.324	0.953
Rule [Dislike [R], Neutral, Like] * Drink [Placebo [R], Alcohol] * Referential Condition [Self [R], Other]						0.588
Rule [Neutral] * Drink [Alcohol] * Referential Condition [Other]				3.796	-4.885 – 12.477	0.391
Rule [Like] * Drink [Alcohol] * Referential Condition [Other]				-0.278	-8.959 – 8.403	0.950
Random Effects						
	Variance	*SD*		*Variance*	*SD*	
Residual	264.78	16.272		264.30	16.257	
ID	14.68	3.831		14.72	3.836	
ICC	0.05			0.05		
N	108 [ID]			108 [ID]		
Observations	1296			1296		
Marginal R^2 / Conditional R^2	0.597 / 0.618			0.598 / 0.619		

Note: Age in years, Gender (Female, Male), Trait Anger Subscale of the STAXi-2, AUDIT Sum, Drink (Placebo, Drink), Referential Condition (Self, Other), Rule (Dislike, Neutral, Like). In model 1, the fixed effects of drink, referential condition and rule were entered into a linear mixed effects model. In model 2, a drink by referential condition by interaction term was entered into a linear mixed effects model. Age, gender, trait anger and AUDIT were also entered into both models as fixed effects to control for their influence. Random effects included random intercepts for subject ID. P values for each fixed effect were estimated using Kenward-Roger d.f.
[R] Reference level for each categorical variable.

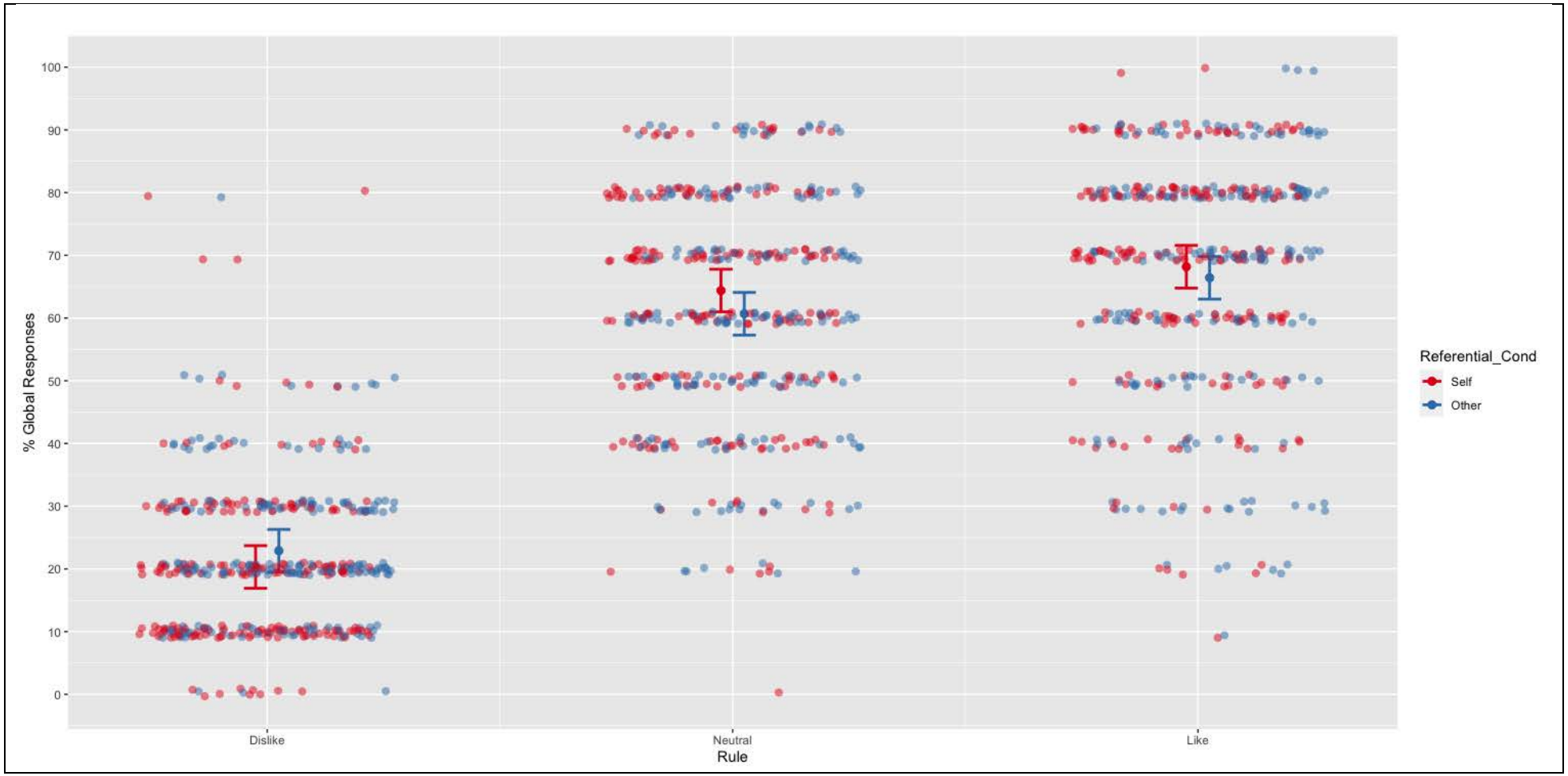

Figure 5.8: % global ratings (i.e., overall learning) in the dislike, neutral and like rule conditions for self and other referential learning. Error bars represent 95% confidence intervals.

5.6. Discussion

This study investigated whether acute alcohol consumption effects hostile evaluations of dyadic social interactions (i.e., two people interacting) in social drinkers, and whether these hostile evaluations were specific to the type of interaction being observed (i.e., non-hostile, ambiguous, hostile). Results show no evidence for an effect of drink on overall hostile ratings of dyadic social interactions. This suggests that social drinkers do not demonstrate an overall hostile attribution bias when observing a dyadic social interaction when intoxicated. This was surprising given that previous research *(see Chapter 4)* concludes greater hostile attribution bias of isolated emotional facial expressions following alcohol. The lack of an effect when seeing dyadic social interaction may be due to the added content and contextual information seen when observing two people interacting. Research suggests that the impressions formed from witnessing third party encounters goes beyond those formed when observing individuals in isolation (Fiske & Haslam, 1996). The proposed Integrative Model of Relational Impression Formation (IMRIF) by Quadflieg and Westmoreland (2019) outlines four psychological attributes that influence impressions formed when observing third party encounters. Content, target and context attributes described in this model are thought to be heavily involved in the accuracy of the impressions formed when observing two people interacting. This additional information may aid the perceiver by providing additional social insights. For example, judging an isolated individual for perceived hostility relies on the accurate interpretation of key facial features that imply greater hostility. Whereas in a dyadic social interaction, there is greater perceptual information provided by the context (i.e., situation circumstances) in which the individuals seen are interacting and the expressive body language displayed (Quadflieg & Penton-Voak, 2017). It is therefore reasonable to speculate that the additional content and contextual information displayed in a dyadic social interaction may dampen

the relative effect of alcohol (compared to placebo) on hostile interpretations. There was also no evidence to suggest that the type of dyadic interaction seen (i.e., non-hostile, ambiguous, hostile) influenced the hostility ratings following alcohol compared to placebo (no drink by dyadic scenario interaction).

These findings are limited by stimuli selection and initial piloting of the dyadic social interaction images used. The criteria for image selection were to include those that displayed a clear image of two individuals interacting in some way. These were then evaluated in a small pilot study to assess the perceived level of hostility displayed in each interaction (to create distinct non-hostile, ambiguous and hostile groupings). This process somewhat oversimplifies the complexity of a social interaction. The literature surrounding third party encounters suggests that impressions of interactions are often made based on social attributes of the encounter. These include interpreting whether two individuals know each other (i.e., strangers, friends, colleagues etc.) (Barnes & Sternberg, 1989; Costanzo & Archer, 1989), the purpose of their interaction (i.e., bonding, disagreeing, problem solving etc.) (Arioli et al., 2018; Canessa et al., 2012), and the type of involvement in the interaction (i.e., rapport, commitment, intimacy etc.) (Bernieri et al., 1996; Fawcett & Gredeback, 2013; Michael et al., 2016). Research also highlights that impressions are influenced by who is thought to have the perceived control/power in an interaction (Mast & Hall, 2004), and which individuals is perceived to be the victim or perpetrator of a particular behaviour (Gray et al., 2014). All of these key features and attributes of a dyadic social interaction arguably influence the degree of hostility perceived and would almost certainly influence the impressions formed following alcohol compared to placebo. Future work should consider this when developing stimuli. More specifically, future research could aim to tease apart the specific attributes of a social interaction by manipulating, for example, whether individuals appear to know each other and the

perceived likeness between two individuals. This would give greater insight into the role that alcohol plays in the impression formation of dyadic social interactions. Finally, the expressive intensity of the emotions displayed by each individual within a dyadic social interaction is likely to influence the impressions formed, and as previously investigated (*see Chapter 2 and Chapter 4)*, acute alcohol influences the recognition of emotional facial expression and the perceived hostility. Future work could adjust the emotions displayed (i.e., angry, happy, sad, disgusted, surprised expressions) and the intensity of the displayed emotions in dyadic social interactions to test whether ambiguity surrounding emotional expressions influences the perceived hostility of a third-party encounter.

This study also investigated whether acute alcohol consumption effects how social drinkers infer social-evaluative information about themselves (i.e., self-referential) and others (i.e., other-referential). As well as, determining whether social-evaluative inferences following alcohol are specific to the self (how evaluations are perceived about themselves) compared to others (how evaluations are perceived towards others). There were two primary outcomes assessed during the social interaction (learning phase) and one primary outcome assessed following it (global learning outcome). During the learning phase, % of positive responses and errors to criterion were used to assess the influence of alcohol on how individuals used feedback to learn whether another person liked them (self-referential)/other (other-referential) during a social interaction (learning phase). Global likeness ratings were used to assess how well they judged whether they/other were liked/disliked following the interaction (global learning outcome). During the learning phase, there was no evidence for a main effect of drink on the % of positive responses. This suggests a similar profile of learning following both alcohol and placebo during a social interaction. There was also no evidence of a drink by rule interaction, suggesting alcohol did not influence how participants

learnt social rules in the dislike, neutral and like conditions (i.e., a similar profile of learning in each rule condition following alcohol compared to placebo). There was no evidence of a drink by referential condition interaction. This suggests that alcohol does not influence how individuals learn social information about perceived likeness towards the self as well as towards others. Collectively, this evidence suggests that acute alcohol consumption does not influence how individuals infer social evaluative information during a social interaction. For errors to criterion scores, there was no evidence for a main effect of drink. This suggests that individuals make similar errors whilst learning the rules (i.e., criterion of 8 consecutive rule congruent responses) following both alcohol and placebo. This supports the claim that a similar profile of learning takes place following both alcohol and placebo drinks. There was evidence for a main effect of rule on errors to criterion scores. Participants made fewer errors when learning the like rule compared to the dislike rule suggesting that individuals are faster at determining whether a social agent likes them/others compared to determining whether they dislike them/others. This is consistent with previous research using a similar self-referential task (Button et al., 2016). These authors report that individuals made fewer errors when learning the self-like rule compared to the self-dislike rule. There was no evidence of a two- or three-way interaction between drink, referential condition and rule conditions. Collectively, this suggests that alcohol consumption does not influence how quickly individuals learn social evaluative information during a social interaction. Specifically, alcohol does not influence errors made before learning a self-like or self-dislike social rule, nor does it influence errors made before learning other-like or other-dislike rules. Following the learning phase, there was no evidence that alcohol influenced the overall perceived likeness rating (i.e., global learning outcome). This suggests that acute alcohol consumption does not influence how social drinkers evaluate whether they (or others) are liked or disliked compared to placebo.

Interestingly, alcohol didn't appear to influence global ratings following the neutral rule (no evidence of an interaction between drink and social rule). Estimates suggest that neutral rule global ratings decreased following alcohol compared to placebo, but this difference was not statistically significant. This result was surprising given the evidence suggesting a difference during the learning phase (i.e., following the neutral rule, alcohol reduced positive evaluations). These results taken together suggest that social drinkers differ in learning the neutral rule following alcohol, but when they reflect on the social interaction there is no difference in the perceived overall likeness demonstrating that the rule was learnt similarly in both alcohol and placebo conditions.

The results from the SELT tested how alcohol influences the way in which social drinkers infer social-evaluative information. This line of enquiry could be extended further to include investigating how other forms of social-evaluative information is inferred. In the context of alcohol related aggression, the SELT task could be adapted in future research to include threatening/hostile vs non-threatening/non-hostile word pairing. Gilman et al. (2008) report that alcohol attenuates response sensitivity to threatening stimuli in part explaining the anti-anxiolytic effects of alcohol. Similarly, acute alcohol consumption has been linked to increased hostile attribution bias of ambiguous social information, leading to increased likelihood of aggressive responding (Bartholow & Heinz, 2006; Subra et al., 2010). It is therefore likely that using an adapted version of the SELT by including threatening and non-threatening word pairings would result in a reduced bias towards evaluating a social agent as threatening following alcohol consumption. It is similarly likely that hostile and non-hostile word pairings would result in an increased bias towards evaluating a social agent as hostile following alcohol.

5.6.1. Conclusions

Findings suggest that social drinkers do not demonstrate a hostile attribution bias when observing a dyadic social interaction following alcohol compared to placebo. These findings were limited by stimuli selection and initial piloting of the dyadic social interaction images used. Future research should tease apart the specific attributes of a social interaction by manipulating whether individuals appear to know each other, and the perceived likeness between two individuals. Future work could also adjust the emotional expression displayed (i.e., angry, happy, sad, disgusted, surprised expressions), and the intensity of the emotions in dyadic social interactions to test whether ambiguity surrounding emotional expressions influences the perceived hostility of third-party encounters. Findings also suggest that social drinkers do not differ in how they process social evaluative information during a social interaction following alcohol and placebo drinks. They also suggest that alcohol does not influence the overall perceived likeness following the social interaction. Future research should explore how other social evaluations are made based on similar cued feedback. Specifically, how threatening/hostile vs non-threatening/non-hostile social feedback from a social agent influences evaluative inferences made following when intoxicated.

CHAPTER 6: GENERAL DISCUSSION

6.1. Chapter Overview

The primary purpose of this thesis of work was to extend the current understanding of the cognitive and perceptual mechanisms of alcohol-related aggression, by investigating the acute and chronic effects of alcohol consumption on several key aspects of social cognition. These aspects included emotional face recognition and hostile attribution bias towards isolated facial expressions, as well as impression formation when observing dyadic social interactions. In addition, approach/avoidance tendencies and social evaluative inferences following alcohol were also explored. It is important to investigate these mechanisms in relation to alcohol-related aggression since the Office for National Statistics (2019) report that 39% of violent incidences were committed by adults under the influence of alcohol (most recent data recorded between April 2017– March 2018). Both observational (non-experimental) and experimental methods have been used to provide evidence on the influence of alcohol consumption on several outcomes. More specifically, the experimental work in this thesis addresses the influence of acute consumption on emotional face processing accuracy in social drinkers (*Chapter 2*), hostile attribution bias towards facial expressions (*Chapter 4*), and hostile attribution bias towards dyadic social interactions (*Chapter 5*). The cross-sectional observational work addresses whether chronic consumption influences emotional face processing (*Chapter 3*). Triangulation of results using these different approaches was implemented to improve the strength of evidence (Heale & Forbes, 2013). Similar effects or associations using similar approaches reduces bias and improves reliability (Lawlor et al., 2016). Comparisons across chapters will therefore be made to improve interpretations.

This final chapter will:

- Summarise the key findings from each study chapter and briefly discuss the independent contribution in relation to the thesis aims
- Triangulate and discuss the findings from all study chapters in relation to previous research
- Evaluate the unique contributions of this work and discuss the implications
- Address limitations of this work and identify areas for future development
- Suggest future lines of inquiry to progress understanding in this field

6.2. Summary of Thesis Findings

Table 6.1 summarises the key research questions and findings of each study chapter. *Chapter 2* found evidence to suggest that alcohol impairs global emotion recognition in social drinkers when compared to placebo. In this chapter there was also evidence of a reduced sensitivity towards sad and fearful expressions following alcohol. These effects were not more pronounced in high compared to low trait aggressive drinkers. In *Chapter 3*, there was evidence to suggest that an increase in chronic alcohol consumption (i.e., the number of units of alcohol consumed per week over a minimum period of 5 years) was associated with a decrease in sadness sensitivity. This suggests that drinking more per week over a longer period of time reduced social drinkers' ability to detect cues of sadness in emotional displays. There was no evidence of an association with global emotion processing deficits. *Chapter 4* found evidence to suggest that ambiguous displays of emotional faces were reported as more hostile following alcohol compared to placebo. When the emotions were less ambiguous (i.e., full examples of the emotion) there was no evidence of a difference in hostility ratings. At an emotion specific level, *Chapter 4* found that happy emotions were rated as more hostile following alcohol. This chapter also found no evidence of an

effect of alcohol on approach/avoidance tendencies when perceiving emotional expressions. *Chapter 5* found no evidence of an effect of alcohol on hostile judgements of dyadic social interaction (i.e., two people interacting), nor was there evidence to suggest that alcohol influenced how social drinkers process social evaluative information during a social interaction.

Table 6.1: Main research questions and findings summarised from each study chapter.

Chapter	Research questions	Research Findings
Chapter 2	Following alcohol compared to placebo: a) Is there evidence of a global deficit in emotion recognition? b) Is there evidence of emotion specific deficits in emotion recognition? c) Are these effects more pronounced in high compared to low trait aggressive drinkers?	a) There was evidence to suggest that acute alcohol consumption reduced global emotion processing accuracy. b) There was also evidence of a reduced sensitivity towards sad and fearful expressions following alcohol. c) There was no evidence to suggest that these effects were more pronounced in high compared to low trait aggressive drinkers
Chapter 3	a) Is there evidence of an association between chronic consumption/binge drinking frequency with impaired global emotion recognition, and is this a global or emotion specific association?	a) There was no evidence of an association for global emotion recognition. There was evidence to suggest an association between increased chronic consumption and a deficit sadness recognition.
Chapter 4	Following alcohol compared to placebo: a) Is there evidence of a hostile attribution bias when social drinkers evaluate emotional facial expression? b) Is there evidence of emotion specific hostile attribution biases? c) Is there evidence to suggest that alcohol influences approach/avoidance tendencies when perceiving emotional facial expressions?	a) There was no evidence of a global hostile attribution bias following alcohol. However, when considering the intensity of the emotion, ambiguous emotions were judged to be more hostile following alcohol. b) There was evidence to suggest that happy emotions were seen as more hostile following alcohol. c) There was no evidence to suggest that alcohol influenced approach/avoidance tendencies when perceiving emotional expressions.
Chapter 5	Following alcohol compared to placebo: a) Is there evidence to suggest that dyadic social interactions are interpreted as more hostile? b) Is there evidence to suggest that social drinkers differ in how they process social evaluative information during a social interaction?	a) There was no evidence of an effect of alcohol on hostile judgements of dyadic social interactions. b) There was no evidence to suggest alcohol influenced how social drinkers process social evaluative information during a social interaction.

6.2.1. Acute Alcohol Consumption Impairs Emotional Face Processing and Specifically Impairs Sad and Fearful Expression Recognition

The primary aim of chapter 2 was to investigate whether emotion processing of facial expressions was affected by acute alcohol consumption in high and low trait aggressive individuals. This was addressed using a double blinded placebo-controlled experiment in which social drinkers completed an emotional face recognition task (presenting the six basic emotional expression: Happy, Angry, Sad, Disgust, Fear, Surprise) following both alcohol and matched placebo drinks. It was hypothesised that there would be a deficit in emotion processing accuracy, as well as an emotion specific increased sensitivity towards perceiving anger, and a decreased sensitivity towards perceiving sadness following alcohol. Results suggest a global deficit in emotion processing accuracy consistent with past research which similarly report poorer emotion recognition following acute alcohol consumption (Tucker & Vuchinich, 1983). The accurate recognition of emotional faces is a key factor involved in successful social interactions (Moriya et al., 2013). In the context of alcohol- related aggression, the reduced ability to accurately identify emotional expressions may contribute to misinterpretation of emotional states and intentions of others, leading to poorer social function when intoxicated (Adolphs & Tusche, 2017). At an emotion specific level, findings suggest a reduced sensitivity towards expressions of sadness and fear, as well as a reduced bias towards seeing happiness following alcohol compared to placebo. There was also weak evidence suggesting reduced sensitivity to disgusted emotional expressions. These results suggest that alcohol differentially impairs the processing of specific emotional facial expressions (i.e., displays of sadness, fear and to a certain extent disgust) which are likely to drive the global deficits in emotion processing. Again, in the context of alcohol-related aggression, these findings have social relevance. Fearful and sad expressions are considered to be signals of distress

and submission (Blair, 2005; Hart, 2011) which can signal avoidance and low confrontation to potential aggressors which, in turn, may curtail aggressive responding. Therefore, a decrease in sensitivity to these emotions following the consumption of alcohol increases the likelihood of aggressive behaviour. A reduced happiness bias following alcohol may function to promote aggressive behaviour, as happy emotions are considered to be a positive (i.e., prosocial) and is often the most easily identifiable emotion (Calvo & Beltran, 2013). The effect of acute alcohol consumption on global and emotion specific processing was not found to be more pronounced in high compared to low trait aggressive individuals.

Chapter 2 also aimed to test whether social drinkers either high or low in trait aggression displayed an emotion processing bias following acute alcohol compared to placebo when the facial stimuli displayed were ambiguous. A forced choice task was used to present emotional expression pairs (i.e., Happy - Angry and Happy - Sad) along a morphed continua transition from a full emotion to another. It was also hypothesised that there would be an increased bias towards angry emotions and a reduced bias towards sad emotions in the 2AFC tasks following alcohol compared to placebo. There was no evidence of alcohol-related bias towards angry faces in the happy-angry morph. This is consistent with Khouja et al. (2019) who similarly report no anger bias in happy-angry facial morphs but contradicts Attwood, Ataya, et al. (2009) who do report an anger bias in negative facial morphs (i.e. anger-disgust facial morphs). Surprisingly, there was evidence, albeit weak, to suggest alcohol led to a sadness perception bias in the happy-sad facial morph. It is unclear whether this captures a reduced happiness or increased sadness perceptual bias. These results are inconsistent with the above reported evidence of a happy bias when tested using an emotion recognition task presenting the six basic emotions (i.e., 6AFC). Therefore, these findings

were interpreted with caution, and further exploration of bias using alternative 2AFC emotion facial morphs (i.e., sad-anger) will help to disentangle this in future research.

6.2.2. Increased Chronic Alcohol Consumption is Associated with Impaired Sadness Recognition

Chapter 3 aimed to extend the findings of Chapter 2 by exploring associations between chronic alcohol consumption and emotional face recognition. The same emotion recognition task used in Chapter 2 measuring global recognition accuracy of the six basic emotions (Happy, Angry, Sad, Disgust, Fear, Surprise), as well as emotion specific response sensitivity and bias was used in this investigation. This cross-sectional study recruited non-dependent drinkers (i.e., regular alcohol consumers without a clinical diagnosis of dependence) that reported consuming alcohol weekly for at least 5 years (in order to capture frequent consumption over time). Information surrounding the typical number of alcoholic drinks consumed per week was collected and used to estimate the number of units consumed per week. Binge drinking frequency was also measured. Similar to the acute alcohol deficits discussed in chapter 2, it was hypothesised that chronic consumption, defined as the number of units consumed per week over a sustained period of at least 5 years, would be associated with poorer global emotion processing accuracy. Similarly, it was anticipated that increased binge drinking would be associated with the same impairment. Associations between units per week and binge drinking frequency with emotion specific response sensitivity and bias was also tested.

There was no evidence of an association between units consumed per week or binge drinking frequency over a 5-year period with global emotion processing accuracy. These findings suggest that chronic alcohol consumption amongst non-dependent drinkers does not appear to impair the ability to accurately recognise emotional facial expressions. Similar research reports a

global emotion processing deficit in alcohol dependent drinkers (Donadon & Osorio, 2017). This suggests global deficits in recognition accuracy may only be present in alcohol dependent samples and the chronicity of alcohol consumption in non-dependent drinkers may not be sufficient enough to produce a deficit. This is supported by evidence from a birth cohort longitudinal study that investigated adolescent and early adulthood (i.e., ages 16-23) binge drinking behaviour on later emotion processing accuracy (i.e., age 24) (Mahedy et al., 2020). Similar to the results in chapter 3, this study reports no association between frequency of binge drinking and global emotion processing accuracy. At an emotion specific level, there was evidence of a reduced sensitivity towards sadness as units of alcohol per week increased. This finding is consistent with similar chronic alcohol consumption and emotion processing research that similarly reports reduced sadness recognition when testing an alcohol dependent sample (Frigerio et al., 2002; Philippot et al., 1999); it is however worth noting this deficit is attributed to an increased misattribution of sad faces as angry, suggesting an anger bias amongst alcohol dependent drinkers. Chapter 3 found no evidence of an anger bias in non-dependent drinkers. This discrepancy is most likely to be due to the different samples used. Non-dependent compared to dependent chronic drinkers differ in baseline aggressive tendencies which may be driving the anger perception bias (Beck & Heinz, 2013). In chronic non-dependent drinkers, a reduced sensitivity towards sadness does however have key alcohol-related aggression implications. As discussed above, sadness is a distress or submission cue (Blair, 2005; Hart, 2011). Therefore, a reduced sensitivity towards seeing this emotion may be a mechanism increasing the likelihood of aggressive responding (as cues of distress and submission are likely to be missed). A reduced sensitivity towards sadness was identified following both acute (i.e., Chapter 2) and chronic (i.e., Chapter 3) alcohol consumption amongst non-dependent drinkers. And again, these findings lend support to previous acute

(Attwood, Ohlson, et al., 2009; Craig et al., 2009) and chronic (Frigerio et al., 2002; Philippot et al., 1999) research that report similar results. Taken together, these strengthen the evidence suggesting that alcohol consumption does in fact disrupt the processing of key social signals of distress and submission (Blair, 2005; Hart, 2011).

The evidence in these chapters (i.e., chapters 2 & 3) highlights a specific impairment to the processing of emotional displays of sadness. The recognition accuracy of this emotion is impaired by acute and chronic consumption. This consistent conclusion drawn from multiple studies in this thesis provide stronger meta-evidence for this emotion specific effect, as triangulating results using different methodologies improves the strength of evidence (Heale & Forbes, 2013).

6.2.3. Acute Alcohol Consumption Increases Hostile Perceptions of Ambiguous Expressions and Happy Faces

Both chapters 2 and 3 focus on emotion processing recognition, that is, how accurate individuals are at identifying the emotion displayed in a facial expression and whether or not biases occur. Chapter 4 extended this line of enquiry by focusing on interpretation of emotional facial expressions following acute alcohol consumption. Specifically, the primary aim of this research was to test hostile attribution bias towards emotional expressions. In addition, a secondary objective aimed to explore approach/avoidance behaviours towards facial expressions. These aims were similarly addressed using a double blinded placebo-controlled experiment in which social drinkers completed a hostile attribution bias task (presenting the six basic emotional expression Happy, Angry, Sad, Disgust, Fear, Surprise) and an approach/avoidance task (presenting Happy, Angry, Sad, Disgust expressions) following both alcohol and matched placebo drinks. It was hypothesised that there would be greater hostile attribution bias towards emotional facial expression following acute alcohol consumption. And for approach/avoidance tendencies, it was

hypothesised that there would be less avoidance of anger and disgusted expressions following acute alcohol.

There was no evidence for an effect of drink on global hostile ratings of emotional expressions suggesting social drinkers do not see facial expressions as more hostile following alcohol. However, when considering emotional intensity, there was evidence to suggest that the emotional intensity displayed influenced hostile interpretations following alcohol compared to placebo. This interaction specifically highlighted that low intensity emotions (i.e., emotionally ambiguous to the perceiver) were rated as more hostile following alcohol. As the intensity of the emotion increased, this alcohol induced difference reduced, resulting in the difference between alcohol and placebo hostility ratings diminishing as a function of intensity. Seeing ambiguous facial expressions as more hostile when intoxicated has social relevance as the propensity to see faces as more hostile may lead to increased aggression and violence (Wegrzyn et al., 2017). Similar past research indicates that higher levels of hostile attribution bias are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006) and consequently plays a role in reactive aggressive behaviour (Crick & Dodge, 1996). Therefore, if individuals see ambiguous facial expressions as more hostile under the influence of alcohol, this may increase the likelihood of aggressive responding. At an emotion specific level, happy facial expressions were seen as more hostile following alcohol compared to placebo. When considering intensity, happy faces were judged as more hostile regardless of the emotional intensity suggesting that even full unambiguous displays of happiness were judged to be more hostile following alcohol. Happy faces being seen as more hostile when intoxicated is of social importance. Calvo and Beltran (2013) suggest that happiness is the most easily recognised expression and is considered to be a positive emotion that promotes prosocial behaviour. Research also demonstrates that increasing the recognition of

happiness in ambiguous emotional expressions results in a reduction in anger and aggressive behaviour (Penton-Voak et al., 2013). Therefore, the increased hostile perception of happy faces following alcohol may increase the likelihood of aggression by reducing the perceivers exposure to these positive cues that promote pro-social behaviour. Results also provide some evidence to suggest that ambiguous displays of anger and disgust were seen as more hostile following alcohol compared to placebo. When factoring emotional intensity, this alcohol induced difference did diminish as the intensity of the emotion increased. This suggests that when facial expressions of anger and disgust are ambiguous to the perceiver, there is an increased tendency to interpret these emotions as hostile following alcohol compared to placebo. But when the display of anger and disgust are clear (i.e., emotionally unambiguous), there is less of an effect of alcohol on hostile attribution bias (seen as similarly hostile following both alcohol and placebo drinks).

Clear comparisons and meta inferences can be made from the conclusions drawn from Chapter 2 (i.e., emotion recognition following acute alcohol) and Chapter 4 (i.e., hostile attribution bias of emotional face following acute alcohol). Chapter 2 demonstrates a global deficit in recognition accuracy of emotional expressions following alcohol. This suggests that alcohol increases the ambiguity of faces by impairing the ability to accurately determine the expressed emotion. Chapter 4 concludes that ambiguous emotional expressions are generally interpreted as more hostile following alcohol compared to placebo and that this difference diminished as intensity increases (i.e., the more unambiguous the expression is, the less hostile attribution bias). Taken together alcohol consumption may result in social drinkers inferring hostile intent when observing others by a) impairing the ability to recognise emotions as effectively, and b) increasing hostile attribution bias of ambiguous emotional facial expressions. And again, these hostile biases are associated with increased aggression (Chen et al., 2012; Crick et al., 2002; Dodge, 2006).

Chapter 4 found that social drinkers show no increased/decreased tendency to approach or avoid an angry or disgusted face following alcohol compared to placebo. The same was also found when exploring approach/avoidance tendencies towards happy/sad expressions following alcohol and placebo. This suggests that acute alcohol consumption does not influence approach/avoidance tendencies towards emotional stimuli. However, these results may be limited by the implicit task demands used. Work is needed to establish whether the tendency to approach or avoid individuals when intoxicated is an implicit (i.e., automatic) or and explicit (i.e., requires evaluation) response (Phaf et al., 2014). As chapter 4 used an implicit measure of approach/avoidance tendencies, future research could conceptually replicate the study using an explicit measure that requires individuals to consciously evaluate the emotional valence of the stimuli before making a decision to approach or avoid (Krieglmeyer & Deutsch, 2010).

6.2.4. Interpretations of Social Interactions are not Influenced by Acute Alcohol Consumption

The first three study chapters focused on the effects of alcohol consumption on isolated emotional face processing. Specifically, chapters 2 and 3 focused on the acute and chronic effects of alcohol consumption on emotional face recognition, respectively. Chapter 4 furthered this enquiry by addressing whether acute alcohol consumption influence hostile attribution biases of emotional facial expressions. Chapter 5 extended this work by testing the effects of acute alcohol on impressions formed when observing third party encounters, as well as social evaluative learning during a social interaction and aimed to address two primary research objectives. Firstly, whether acute alcohol consumption effects hostile evaluations of dyadic social interactions (i.e., two people interacting) in social drinkers. In addition, secondary objectives include exploring whether hostile perception of dyadic interactions differs when viewing different types of interaction (i.e., non-

hostile, ambiguous, hostile) following alcohol consumption. It was hypothesised that dyadic social interactions will be seen as more hostile following acute alcohol consumption compared to placebo. This effect is anticipated to be more pronounced in ambiguous interactions. The second objective of this chapter aimed to investigate whether alcohol consumption effects how social drinkers infer social-evaluative information about the self (self-referential) and others (other-referential). It was hypothesised that, following alcohol, social drinkers will be poorer at using cued feedback during the learning phase of a social interaction resulting in increased perceived negative evaluation. Similarly, it was hypothesised that overall perceived likeness following the social interaction will be lower following alcohol compared to placebo.

For impressions formed when observing dyadic social interactions, results show no evidence for a difference in hostile ratings of dyadic social interactions following alcohol compared to placebo. Specifically, ambiguous dyadic stimuli were not seen as more hostile following alcohol. This was surprising given previous research that suggests that ambiguous social stimuli are often interpreted as more hostile (Milich & Dodge, 1984). For example, research suggests that ambiguous social interactions are typically reported to be perceived as more hostile following alcohol consumption (Nasby et al., 1980). This work however concludes that alcohol influences hostile interpretations of ambiguous interactions in typically aggressive individuals. The inconsistency in findings between those reported in chapter 5 and this research may therefore be a result of the unselected sample used (i.e., typical non-aggressive social drinkers). Future work should address this by recruiting high trait aggressive individuals specifically to see if this individual difference influences the anticipated effect of alcohol on hostile attribution bias when perceiving dyadic interactions. Similarly, Quadflieg and Westmoreland (2019) proposed the Integrative Model of Relational Impression Formation (IMRIF) which outlines key attributes that

influence impression formation when viewing dyadic social interactions. One of which is situational context that provides individuals with additional perceptual information that helps impression formation. It is therefore reasonable to speculate that the additional content and contextual information displayed in a dyadic social interaction may dampen the relative effect of alcohol (compared to placebo) on hostile interpretations. To establish this, future research could manipulate the contextual information provided to test whether this does in fact influence hostile judgements, particularly when the contextual information is ambiguous.

For social evaluation processing during a social interaction, findings suggest a similar profile of learning following both alcohol and placebo (i.e., no evidence of a main effect of drink nor drink by rule/referential condition interaction for both learning phase outcome measures). This suggests that social drinkers similarly learn whether they (or others) were liked or disliked following feedback from a social agent following alcohol and placebo drinks. This task also measured whether individuals were able to determine if they (or others) were liked or disliked following the social interaction (i.e., global likeness rating after the interaction took place). Findings suggest no evidence that alcohol influenced the overall perceived likeness rating (i.e., global learning outcome). This suggests that acute alcohol consumption does not influence how social drinkers evaluate whether they (or others) are liked or disliked compared to placebo after the interaction.

6.3. Original Research Contributions and Implications

There is a wealth of quantitative and qualitative evidence that highlights a link between alcohol consumption (both acutely and chronically consumed) and aggression (Beck & Heinz, 2013; Bushman & Cooper, 1990; Chermack & Giancola, 1997; Hoaken & Stewart, 2003; Ito et al., 1996; Lipsey et al., 1997). Several cognitive mechanisms that increase the likelihood of alcohol-

related aggression have been previously discussed as potential influential factors involved in this causal relationship. This thesis of work contributes to this line of inquiry by establishing and exploring other potential mechanisms that contribute to this increased likelihood of responding aggressively following alcohol. This work uniquely explains the role of emotional face processing and social judgements of perceptual information in increasing the propensity towards aggression. Though speculative, it argues that key perceptual information displayed during a social interaction plays a functional role in aggressive behaviour. However, it does not conclude that this mechanism is the sole contributing factor. Social interactions are complex, and several behavioural mechanisms are at play. It is well documented that alcohol impairs behavioural control (i.e., response activation and inhibition). Experimental evidence concludes that this makes drinkers more likely to fail to suppress maladaptive behaviours and therefore increases the likelihood of aggressive behaviour (de Wit et al., 2000; Marczinski et al., 2005). This current thesis of work argues that alcohol impairs the ability to recognise emotional facial expressions globally and important social cues of submission and distress (i.e., sad facial expressions). This, coupled with prior knowledge of impaired behaviour control, may further explain why individuals react aggressively (i.e., failure to suppress a response triggered by poor processing of social cues). This highlights the complex interplay between alcohol induced deficits in behaviour control and the processing of socially relevant information.

Another well documented theory of alcohol-related aggression suggests that alcohol produces a myopic effect. Steele and Josephs (1990) argue that alcohol intoxication impairs cognitive processes and influences aggressive behaviour by narrowing attentional focus. It creates a myopic effect in which attention can only focus on the most salient and easily processed cues within a social environment, resulting in key social cues being missed. In the context of emotional

expressions, this theory suggests that only the most salient cues within a social interaction would be attended to and processed. This thesis concluded that when considering low emotional intensity facial expressions (i.e., emotionally ambiguous), faces were perceived as more hostile which in turn has an adaptive role in aggressive behaviour. Although tentative, this could be explained by the alcohol myopia theory, as attention focuses on the most salient cues whilst intoxicated, individuals could focus on cues of threat or danger as it is adaptively better to use resources to identify these cues. Alcohol consumption may therefore lead to benign ambiguous social cues being interpreted as hostile.

6.4. Limitations

The acute alcohol studies in this thesis had a relatively low placebo manipulation success rate. This may have been a direct consequence of asking the participants to retrospectively judge whether they believed they had consumed alcohol or not at the end of the testing session. A post-consumption decision of having received alcohol is likely to be influenced by post-ingestion consequences of drink (i.e., whether they received alcohol or not). The relative influence (and therefore accuracy) of that will increase over time as they have longer to experience these effects. Post-session judgements were used to measure the relative success of the placebo manipulation. However, this proxy could have been directly influenced by time (i.e., allowing the participant to experience the effects of alcohol, or lack thereof) or the anticipated effects not meeting expectations (i.e., initially judging to have received alcohol and changing this judgement when anticipated expectations were not met). Nevertheless, there appeared to be a limiting lack of control over the anticipated effects of alcohol. Evidence has shown that the expectation of alcohol leads to individuals adapting their behaviour to compensate for the anticipated effects of alcohol (Marczinski & Fillmore, 2005). Therefore, those that expected alcohol may have adapted their

behaviour to compensate for the anticipated effects which may have dampened the observed effects of alcohol in the retrospective studies. Future emotion processing and dyadic social interaction interpretation research could specifically test the influence of alcohol expectation, and the direct pharmacological influence of the drug using a balanced placebo design (Sayette et al., 1994). This design would allow an anti-placebo (i.e., alcohol administered but not expected) vs. control (i.e., no alcohol administered and not expected) comparison which best models a pure pharmacological effect. It would also allow effects that are due to expectancy to be tested (i.e., placebo vs. control).

The chronic alcohol consumption research in this thesis is limited by the cross-sectional nature in which it was conducted. This design only allows the examination of associations at one time point making causal inferences difficult (Setia, 2016). Longitudinal studies also have the distinct advantage of identifying and relating events to a particular exposure (i.e., alcohol consumption) and to further define these exposures with regards to chronicity (i.e., continued consumption over time) (Caruana et al., 2015). Future chronic alcohol work should aim to use a longitudinal approach when exploring the influence of early chronic drinking behaviour and the influence this exposure has on later emotion processing deficits. There is some initial work in the literature that use longitudinal approaches to test this. Research exploring the Avon Longitudinal Study of Parents and Children (ALSPAC) birth cohort data aimed to establish whether observed chronic drinking during adolescence/early adulthood predicts poorer emotion processing in adulthood. Mahedy et al. (2020) tested the influence that binge drinking between the ages of 16-23 had on cognitive measures of working memory, response inhibition and emotional face processing at age 24. They concluded that adolescent and early adulthood binge drinking was not associated with later global emotion processing deficits. To build on this, future research could explore the influence of chronic consumption on emotion specific response sensitivity as well as

response bias to investigate whether alcohol consumption over time is associated with impairments at an emotion specific rather than global only.

6.5. Future Directions

There is strong meta evidence to suggest that facial signals of submission and distress are impaired by acute and chronic consumption. There is also good evidence to suggest that impaired emotion processing can be retrained (Penton-Voak et al., 2013). This work specifically promoted happiness perception when emotional displays were ambiguous and in turn reduced aggressive behaviour. It is therefore plausible that alcohol induced perceptual deficits (i.e., reduced sadness sensitivity) could be retrained. This may be particularly difficult to implement in acute alcohol consumption research (i.e., the influence a single dose exposure has on emotion process) but could be tested on a non-dependent sample of chronic drinkers. Promoting more accurate emotion recognition (i.e., feedback to help individuals learn the correct emotions displayed) in chronic non-dependent drinkers may function to reduce alcohol-related aggression as it could make individuals more aware of the key signals of distress and submission (i.e., more accurate recognition of sadness). Similarly, results from this thesis suggest that happiness is perceived as more hostile following acute alcohol consumption (both ambiguous and full exemplar displays). A similar method of intervention could be adopted to reduce hostile attribution bias of this positive emotion. Recent research has demonstrated that hostile attribution biases can be modified in a sample of adolescents (Van Bockstaele et al., 2020). These authors specifically trained individuals to make more benign interpretations of ambiguously provocative social situations. They concluded that this retraining method reduced biases and also reduced proactive aggressive tendencies. A similar model of training could be implemented in adult drinkers to encourage ambiguous social situations to be perceived as more benign in an attempt to reduce hostile interpretations. This preliminary

idea would initially need validating by testing whether adult hostile interpretations of socially relevant information could in fact be retrained. Specifically, simple feedback could be provided to the participant (i.e., correct or incorrect) during the hostile attribution bias task to lower hostile evaluations.

The final study chapter of this thesis of work aimed to explore the influence alcohol consumption had on processing dyadic social information. This work is in its infancy and future work should aim to build upon this. Dyadic social interactions are complex, and it is likely that the lack of an alcohol effect in this thesis may be due to the oversimplification of these. The literature surrounding this suggests that impressions of interactions are often made based on social attributes of the encounter. These include interpreting whether two individuals know each other (i.e., strangers, friends, colleagues etc.) (Barnes & Sternberg, 1989; Costanzo & Archer, 1989), the purpose of their interaction (i.e., bonding, disagreeing, problem solving etc.) (Arioli et al., 2018; Canessa et al., 2012), and the type of involvement in the interaction (i.e., rapport, commitment, intimacy etc.) (Bernieri et al., 1996; Fawcett & Gredeback, 2013; Michael et al., 2016). These key features of a dyadic social interaction arguably influence the degree of hostility perceived and would almost certainly influence the impressions formed following alcohol compared to placebo. Future research could aim to tease apart the specific attributes of a social interaction by manipulating, for example, whether individuals appear to know each other and the perceived likeness between two individuals. This would give greater insight into the role that alcohol plays in the impression formation of dyadic social interactions.

6.6. Thesis Conclusions

This thesis of work aimed to establish whether acute and chronic alcohol consumption influenced the processing of socially relevant information. Specifically, it tested how acute alcohol

consumption influenced the recognition accuracy and hostile perceptions of isolated facial expression, as well as dyadic social interactions. It also tested how chronic alcohol consumption was associated with emotion recognition in social drinkers. Findings suggest that acute alcohol consumption disrupts the recognition of emotional facial expressions (i.e., chapter 2). They also suggest that when emotional expressions are ambiguous, individuals perceive these to be more hostile (i.e., chapter 4). The global recognition deficit paired with the increased hostile perception when ambiguous may lead to maladaptive behaviour. At an emotion specific level, acute alcohol decreases the ability to detect distress and submissive social cues, such as sad and fearful emotional expressions (i.e., chapter 2). In addition, findings indicate that happy emotions are seen as more hostile following alcohol (i.e., chapter 4). Taken together, acute alcohol may increase the likelihood of aggression by diminishing the ability to see signals of distress and submission and by increasing hostile attribution bias of happiness (an emotion linked to pro-sociability). Chronic consumption seems to be associated with a similar sadness recognition deficit specifically rather than a global deficit as seen in the acute consumption work (i.e., chapter 3). The association found between increased chronic alcohol consumption and a reduced sadness sensitivity is important considering this emotion has been found to curtail aggression. Key meta-inferences can be made when considering the impact chronic and acute consumption has on the processing of isolated emotional expressions. Sadness perception seems to be influenced by both chronic and acute consumption. As this emotion signals distress, it may function to promote aggressive responding. Similarly, happiness was interpreted as more hostile following alcohol. This positive emotion often promotes pro-sociability and seeing it as more hostile when intoxicated may also increase the likelihood of aggressive responding. Future research could investigate whether these deficits in emotional face processing can be retrained. This thesis also extended this line of inquiry by investigating the

effects of alcohol on the processing of dyadic social interactions and findings suggest that social drinkers do not demonstrate a hostile attribution bias when observing these. These findings were limited by stimuli selection and initial piloting of the dyadic social interaction images used. Future work could also adjust the emotional expression displayed (i.e., angry, happy, sad, disgusted, surprised expressions), and the intensity of the emotions in dyadic social interactions to test whether ambiguity surrounding emotional expressions influences the perceived hostility of third-party encounters. Chronic alcohol consumption and emotion face processing findings were limited by the cross-sectional design used. Future research should assess associations between chronic alcohol consumption and emotional face processing longitudinally. This could involve using momentary assessment technology that collects high temporal density drinking data as the event occurs reducing recall bias. In addition, future work could investigate this topic using birth cohort data capturing drinking behaviour across a prolonged period of time.

LIST OF ABBREVIATIONS

2AFC – Two-Alternative Forced Choice Task

6AFC – Six-Alternative Forced Choice Task

AAT – Approach Avoidance Task

ABV – Alcohol by volume

ADHD – Attention Deficit Hyperactivity Disorder

ALSPAC – Avon Longitudinal Study of Parents and Children

ANOVA – Analysis of Variance

AUDIT – Alcohol Use Disorders Identification Test

AXi – Anger Expression Index Subscale of the STAXI-2

BAES – Biphasic Alcohol Effect Scale

BFNE-II – Brief Fear of Negative Evaluations – II

BrAC – Breath Alcohol Concentration

CI – 95% Confidence Intervals

DOI – Digital Object Identifier

EEG – Electroencephalogram

FA – False Alarm

GAD – General Aggression Model

H – Hit Rate

HAB – Hostile Attribution Bias

HABT – Hostile Attribution Bias Task

HABT-D– Dyadic Hostile Attribution Bias Task

HIBT – Hostile Interpretation Bias Task

ICC – Intraclass Correlation Coefficient

ID – Identification

LME – Linear Mixed Effects

M – Mean

MRI – Magnetic Resonance Imaging

NHS – United Kingdom National Health Service

OFC – Orbitofrontal Cortex

OSF – Open Science Framework

PANAS – Positive & Negative Affect Scale

PHQ-9 – Patient Health Questionnaire – 9

RT – Reaction Time

S-Ang – State Anger Subscale of the STAXI-2

SD – Standard Deviation

SDT – Signal Detection Theory

SELT – Social Evaluation Learning Task

SES – Socioeconomic Status

STAXI-2 – State-Trait Anger Expression Inventory

T-Ang – Trait Anger Subscale of the STAXI-2

TARG – Tobacco & Alcohol Research Group

UK – United Kingdom

REFERENCE LIST

Abroms, B. D., Fillmore, M. T., & Marczinski, C. A. (2003). Alcohol-induced impairment of behavioral control: Effects on the alteration and suppression of prepotent responses. *Journal of Studies on Alcohol, 64*(5), 687-695.

Adolphs, R. (2002). Neural systems for recognizing emotion. *Current Opinion in Neurobiology, 12*(2), 169-177.

Adolphs, R., & Tranel, D. (2000). Emotion recognition and the human amygdala. In J. P. Aggleton (Ed.), *The Amygdala. A Functional Analysis.* (pp. 587-630). Oxford University Press.

Adolphs, R., Tranel, D., Hamann, S., Young, A. W., Calder, A. J., Phelps, E. A., Anderson, A., Lee, G. P., & Damasio, A. R. (1999). Recognition of facial emotion in nine individuals with bilateral amygdala damage. *Neuropsychologia, 37*(10), 1111-1117.

Adolphs, R., & Tusche, A. (2017). From Faces to Prosocial Behavior: Cues, Tools, and Mechanisms. *Current Directions in Psychological Science, 26*(3), 282-287.

Anderson, A. K., Spencer, D. D., Fulbright, R. K., & Phelps, E. A. (2000). Contribution of the anteromedial temporal lobes to the evaluation of facial emotion. *Neuropsychology, 14*(4), 526-536.

Anderson, C. A., & Bushman, B. J. (2002). Human aggression. *Annual Review of Psychology, 53*, 27-51.

Archer, J. (2004). Sex Differences in Aggression in Real-World Settings: A Meta-Analytical Review. *Review of General Psychology, 8*, 291-322.

Archer, J. (2006). Testosterone and human aggression: an evaluation of the challenge hypothesis. *Neuroscience & Biobehavioral Reviews, 30*(3), 319-345.

Arioli, M., Perani, D., Cappa, S., Proverbio, A. M., Zani, A., Falini, A., & Canessa, N. (2018). Affective and cooperative social interactions modulate effective connectivity within and between the mirror and mentalizing systems. *Human Brain Mapping, 39*(3), 1412-1427.

Attwood, A. S., Ataya, A. F., Benton, C. P., Penton-Voak, I. S., & Munafo, M. R. (2009). Effects of alcohol consumption and alcohol expectancy on the categorisation of perceptual cues of emotional expression. *Psychopharmacology, 204*(2), 327-334.

Attwood, A. S., Easey, K. E., Dalili, M. N., Skinner, A. L., Woods, A., Crick, L., Ilett, E., Penton-Voak, I. S., & Munafo, M. R. (2017). State anxiety and emotional face recognition in healthy volunteers. *Royal Society Open Science, 4*(5), 160855.

Attwood, A. S., & Munafo, M. R. (2014). Effects of acute alcohol consumption and processing of emotion in faces: Implications for understanding alcohol-related aggression. *Journal of Psychopharmacology, 28*(8), 719-732.

Attwood, A. S., Ohlson, C., Benton, C. P., Penton-Voak, I. S., & Munafo, M. R. (2009). Effects of acute alcohol consumption on processing of perceptual cues of emotional expression. *Journal of Psychopharmacology, 23*(1), 23-30.

Baayen, R. H., Davidson, D. J., & Bates, D. M. (2008). Mixed-effects modeling with crossed random effects for subjects and items. *Journal of Memory and Language, 59*(4).

Bailey, D. S., & Taylor, S. P. (1991). Effects of Alcohol and Aggressive Disposition on Human Physical Aggression. *Journal of Research in Personality, 25*(3), 334-342.

Bamford, S., Penton-Voak, I., Pinkney, V., Baldwin, D. S., Munafo, M. R., & Garner, M. (2015). Early effects of duloxetine on emotion recognition in healthy volunteers. *Journal of Psychopharmacology, 29*(5), 634-641.

Bandura, A. (1973). *Aggression: A social learning analysis*. Prentice Hall.

Bandura, A. (2001). Social cognitive theory: an agentic perspective. *Annual Review of Psychology, 52*, 1-26.

Barbas, H., Saha, S., Rempel-Clower, N., & Ghashghaei, T. (2003). Serial pathways from primate prefrontal cortex to autonomic areas may influence emotional expression. *BMC Neuroscience, 4*, 25.

Barnes, M. L., & Sternberg, R. J. (1989). Social intelligence and decoding of nonverbal cues. *Intelligence, 13*, 263–287.

Baron, R. A., & Richardson, D. R. (1994). *Human aggression* (2nd ed. ed.). Plenum Press.

Bartholow, B. D., & Heinz, A. (2006). Alcohol and aggression without consumption. Alcohol cues, aggressive thoughts, and hostile perception bias. *Psychological Science, 17*(1), 30-37.

Bartholow, B. D., Pearson, M., Sher, K. J., Wieman, L. C., Fabiani, M., & Gratton, G. (2003). Effects of alcohol consumption and alcohol susceptibility on cognition: a psychophysiological examination. *Biological Psychology, 64*(1-2), 167-190.

Bates, D., Maechler, M., Bolker, B., & Walker, S. (2015). Fitting Linear Mixed-Effects Models Using lme4. *Journal of Statistical Software, 67*(1), 1-48.

Bates, M. E., Bowden, S. C., & Barry, D. (2002). Neurocognitive impairment associated with alcohol use disorders: implications for treatment. *Experimental and Clinical Psychopharmacology, 10*(3), 193-212.

Batrinos, M. L. (2012). Testosterone and aggressive behavior in man. *International Journal of Endocrinology, 10*(3), 563-568.

Beaver, J. D., Lawrence, A. D., Passamonti, L., & Calder, A. J. (2008). Appetitive motivation predicts the neural response to facial signals of aggression. *Journal of Neuroscience Research, 28*(11), 2719-2725.

Beck, A., & Heinz, A. (2013). Alcohol-related aggression-social and neurobiological factors. *Deutsches Aerzteblatt International, 110*(42), 711-715.

Berkowitz, L. (1989). Frustration-aggression hypothesis: examination and reformulation. *Psychological Bulletin, 106*(1), 59-73.

Berkowitz, L. (1990). On the formation and regulation of anger and aggression. A cognitive-neoassociationistic analysis. *American Psychologist, 45*(4), 494-503.

Berkowitz, L. (1993). *Aggression: Its causes, consequences, and control.* McGraw-Hill.

Bernieri, F. J., & Gillis, J. S. (1995). The judgment of rapport: A cross-cultural comparison between Americans and Greeks. *Journal of Nonverbal Behavior, 19*, 115-130.

Bernieri, F. J., Gillis, J. S., Davis, J. M., & Grahe, J. E. (1996). Dyad rapport and the accuracy of its judgment across situations: A lens model analysis. *Journal of Personality and Social Psychology, 71*, 110–129.

Best, M., Williams, J. M., & Coccaro, E. F. (2002). Evidence for a dysfunctional prefrontal circuit in patients with an impulsive aggressive disorder. *Proc Natl Acad Sci U S A, 99*(12), 8448-8453.

Birkley, E. L., & Eckhardt, C. I. (2015). Anger, hostility, internalizing negative emotions, and intimate partner violence perpetration: A meta-analytic review. *Clinical Psychology Review, 37*, 40-56.

Blair, R. J. (2003). Facial expressions, their communicatory functions and neuro-cognitive substrates. *Philosophical Transactions of the Royal Society B: Biological Sciences, 358*(1431), 561-572.

Blair, R. J. (2005). Applying a cognitive neuroscience perspective to the disorder of psychopathy. *Development and Psychopathology, 17*(3), 865-891.

Blair, R. J., Colledge, E., Murray, L., & Mitchell, D. G. (2001). A selective impairment in the processing of sad and fearful expressions in children with psychopathic tendencies. *Journal of Abnormal Child Psychology, 29*(6), 491-498.

Blair, R. J., Morris, J. S., Frith, C. D., Perrett, D. I., & Dolan, R. J. (1999). Dissociable neural responses to facial expressions of sadness and anger. *Brain, 122 (Pt 5)*, 883-893.

Blume, A. W., & Guttu, B. L. (2015). Categories of alcohol outcome expectancies and their relationships to alcohol related consequences. *Addictive Behaviors Reports, 1*, 64-67.

Bora, E., & Zorlu, N. (2017). Social cognition in alcohol use disorder: a meta-analysis. *Addiction, 112*(1), 40-48.

Bossuyt, E., Moors, A., & De Houwer, J. (2014). On angry approach and fearful avoidance: The goal-dependent nature of emotional approach and avoidance tendencies. *Journal of Experimental Social Psychology, 50*, 118-124.

Breiter, H. C., Etcoff, N. L., Whalen, P. J., Kennedy, W. A., Rauch, S. L., Buckner, R. L., Strauss, M. M., Hyman, S. E., & Rosen, B. R. (1996). Response and habituation of the human amygdala during visual processing of facial expression. *Neuron, 17*(5), 875-887.

Bushman, B. J. (1997). Effects of alcohol on human aggression. Validity of proposed explanations. *Recent Developments in Alcoholism, 13*, 227-243.

Bushman, B. J., & Cooper, H. M. (1990). Effects of Alcohol on Human Aggression: An Integrative Research Review. *Psychological Bulletin, 107*(3), 341-354.

Buss, A. H. (1961). *The psychology of aggression*. Wiley.

Buss, A. H., & Perry, M. (1992). The aggression questionnaire. *Journal of Personality and Social Psychology, 63*(3), 452-459.

Button, K. S., Browning, M., Munafo, M. R., & Lewis, G. (2012). Social inference and social anxiety: evidence of a fear-congruent self-referential learning bias. *Journal of Behavior Therapy and Experimental Psychiatry, 43*(4), 1082-1087.

Button, K. S., Karwatowska, L., Kounali, D., Munafo, M. R., & Attwood, A. S. (2016). Acute anxiety and social inference: An experimental manipulation with 7.5% carbon dioxide inhalation. *Journal of Psychopharmacology, 30*(10), 1036-1046.

Button, K. S., Kounali, D., Stapinski, L., Rapee, R. M., Lewis, G., & Munafo, M. R. (2015). Fear of negative evaluation biases social evaluation inference: evidence from a probabilistic learning task. *PLoS One, 10*(4), e0119456.

Bye, E. K. (2007). Alcohol and violence: use of possible confounders in a time-series analysis. *Addiction, 102*(3), 369-376.

Calvo, M. G., & Beltran, D. (2013). Recognition advantage of happy faces: Tracing the neurocognitive processes. *Neuropsychologia, 51*(11), 2051-2060.

Canessa, N., Alemanno, F., Riva, F., Zani, A., Proverbio, A. M., Mannara, N., Perani, D., & Cappa, S. F. (2012). The neural bases of social intention understanding: the role of interaction goals. *PLoS One, 7*(7), e42347.

Capito, E. S., Lautenbacher, S., & Horn-Hofmann, C. (2017). Acute alcohol effects on facial expressions of emotions in social drinkers: a systematic review. *Psychology Research and Behavior Management, 10*, 369-385.

Caruana, E. J., Roman, M., Hernandez-Sanchez, J., & Solli, P. (2015). Longitudinal studies. *Journal of Thoracic Disease, 7*(11), E537-540.

Casbon, T. S., Curtin, J. J., Lang, A. R., & Patrick, C. J. (2003). Deleterious effects of alcohol intoxication: diminished cognitive control and its behavioral consequences. *Journal of Abnormal Psychology, 112*(3), 476-487.

Chapman, H., Gillespie, S. M., & Mitchel, I. J. (2018). Facial affect processing in incarcerated violent males: A systematic review. *Aggression and Violent Behavior, 38*, 123-138.

Chen, M., & Bargh, J. A. (1999). Consequences of automatic evaluation: immediate behavioral predispositions to approach or avoid the stimulus. *Personality and Social Psychology Bulletin, 25*, 215–224.

Chen, P., Coccaro, E. F., & Jacobson, K. C. (2012). Hostile attributional bias, negative emotional responding, and aggression in adults: moderating effects of gender and impulsivity. *Aggressive Behavior, 38*(1), 47-63.

Chermack, S. T., & Giancola, P. R. (1997). The relation between alcohol and aggression: An integrated biopsychosocial conceptualization. *Clinical Psychology Review, 17*(6), 621-649.

Coccaro, E. F., McCloskey, M. S., Fitzgerald, D. A., & Phan, K. L. (2007). Amygdala and orbitofrontal reactivity to social threat in individuals with impulsive aggression. *Biological Psychiatry, 62*(2), 168-178.

Cogan, R., & Ballinger, B. C., 3rd. (2006). Alcohol problems and the differentiation of partner, stranger, and general violence. *Journal of Interpersonal Violence, 21*(7), 924-935.

Cohen, J. (1988). *Statistical power analysis for the behavioral sciences*. Erlbaum.

Collins, J. J., Schlenger, W. E., & Jordan, B. K. (1988). Antisocial personality and substance abuse disorders. *The Bulletin of the American Academy of Psychiatry and the Law, 16*(2), 187-198.

Cooper, M. L., Frone, M. R., Russell, M., & Mudar, P. (1995). Drinking to regulate positive and negative emotions: a motivational model of alcohol use. *Journal of Personality and Social Psychology, 69*(5), 990-1005.

Costanzo, M., & Archer, D. (1989). Interpreting the expressive behavior of others: the interpersonal perception task. *Journal of Nonverbal Behavior, 13*, 225–244.

Cox, W. M., & Klinger, E. (1990). Incentive motivation, affective change, and alcohol use: a model. In W. M. Cox (Ed.), *Why People Drink* (pp. 291–314). Gardner Press.

Craig, L. C., Attwood, A. S., Benton, C. P., Penton-Voak, I. S., & Munafo, M. R. (2009). Effects of acute alcohol consumption and alcohol expectancy on processing of perceptual cues of emotional expression. *Journal of Psychopharmacology, 23*(3), 258-265.

Crick, N. R., & Dodge, K. A. (1996). Social information-processing mechanisms in reactive and proactive aggression. *Child development, 67*(3), 993-1002.

Crick, N. R., Grotpeter, J. K., & Bigbee, M. A. (2002). Relationally and physically aggressive children's intent attributions and feelings of distress for relational and instrumental peer provocations. *Child development, 73*(4), 1134-1142.

Dahiru, T. (2008). P - value, a true test of statistical significance? A cautionary note. *Ann Ib Postgrad Med, 6*(1), 21-26.

Darke, S. (2010). The toxicology of homicide offenders and victims: A review. *Drug and Alcohol Review, 29*(2), 202-215.

Davidson, R. J., Putnam, K. M., & Larson, C. L. (2000). Dysfunction in the neural circuitry of emotion regulation--a possible prelude to violence. *Science, 289*(5479), 591-594.

de Gelder, B. (2006). Towards the neurobiology of emotional body language. *Nature Reviews Neuroscience, 7*(3), 242-249.

de Wit, H. (2009). Impulsivity as a determinant and consequence of drug use: a review of underlying processes. *Addiction Biology, 14*, 22-31.

de Wit, H., Crean, J., & Richards, J. B. (2000). Effects of d-amphetamine and ethanol on a measure of behavioral inhibition in humans. *Behavioral Neuroscience, 114*(4), 830-837.

Deffenbacher, J. L. (2000). Prisoners of hate: The cognitive basis of anger, hostility and violence. *Journal of Cognitive Psychotherapy, 14*(2), 201.

Demenescu, L. R., Kortekaas, R., den Boer, J. A., & Aleman, A. (2010). Impaired attribution of emotion to facial expressions in anxiety and major depression. *PLoS One, 5*(12), e15058.

Denson, T. F., Capper, M. M., Oaten, M., Friese, M., & Schofield, T. P. (2011). Self-control training decreases aggression in response to provocation in aggressive individuals. *Journal of Research in Personality, 45*, 252-256.

Dermen, K. H., & George, W. H. (1989). Alcohol expectancy and the relationship between drinking and physical aggression. *The Journal of Psychology, 123*(2), 153-161.

Derntl, B., Windischberger, C., Robinson, S., Kryspin-Exner, I., Gur, R. C., Moser, E., & Habel, U. (2009). Amygdala activity to fear and anger in healthy young males is associated with testosterone. *Psychoneuroendocrinology, 34*(5), 687-693.

DeWall, C. N., Baumeister, R. F., Stillman, T. F., & Gailliot, M. T. (2007). Violence restrained: Effects of self-regulation and its depletion on aggression. *Journal of Experimental Social Psychology, 43*, 62-76.

Dodge, K. A. (2006). Translational science in action: hostile attributional style and the development of aggressive behavior problems. *Development and Psychopathology, 18*(3), 791-814.

Dolder, P. C., Holze, F., Liakoni, E., Harder, S., Schmid, Y., & Liechti, M. E. (2017). Alcohol acutely enhances decoding of positive emotions and emotional concern for positive stimuli and facilitates the viewing of sexual images. *Psychopharmacology, 234*(1), 41-51.

Dollard, J., Doob, L. W., Miller, N. E., Mowrer, O. H., & Sears, R. R. (1939). *Frustrations and aggression*. Yale University Press.

Donadon, M. F., & Osorio, F. D. (2017). Current alcohol dependence and emotional facial expression recognition: a cross-sectional study. *Archives of Clinical Psychiatry, 44*(3), 56-62.

Donadon, M. F., & Osorio Fde, L. (2014). Recognition of facial expressions by alcoholic patients: a systematic literature review. *Neuropsychiatric Disease and Treatment, 10*, 1655-1663.

Duke, A. A., Smith, K. M. Z., Oberleitner, L. M. S., Westphal, A., & McKee, S. A. (2018). Alcohol, drugs, and violence: A meta-meta-analysis. . *Psychology of Violence, 8*(2), 238–249.

Eastwood, A. P., Penton-Voak, I. S., Munafo, M. R., & Attwood, A. S. (2020). Effects of acute alcohol consumption on emotion recognition in high and low trait aggressive drinkers. *Journal of Psychopharmacology*, 269881120922951.

Eckhardt, C. I., & Cohen, D. J. (1997). Attention to anger-relevant and irrelevant stimuli following naturalistic insult. *Personality and Individual Differences, 23*, 619-629.

Eckhardt, C. I., & Crane, C. (2008). Effects of alcohol intoxication and aggressivity on aggressive verbalizations during anger arousal. *Aggressive Behavior, 34*(4), 428-436.

Eckhardt, C. I., Norlander, B., & Deffenbacher, J. L. (2004). The assessment of anger and hostility: a critical review. *Aggression and Violent Behavior, 9*(1), 17-43.

Eisenberg, N., Fabes, R. A., Miller, P. A., Fultz, J., Shell, R., Mathy, R. M., & Reno, R. R. (1989). Relation of sympathy and personal distress to prosocial behavior: a multimethod study. *Journal of Personality and Social Psychology, 57*(1), 55-66.

Ekman, P. (1992). An Argument for Basic Emotions. *Cognition & Emotion, 6*(3-4), 169-200.

Enter, D., Spinhoven, P., & Roelofs, K. (2014). Alleviating social avoidance: effects of single dose testosterone administration on approach-avoidance action. *Hormones and Behavior, 65*(4), 351-354.

Fawcett, C., & Gredeback, G. (2013). Infants use social context to bind actions into a collaborative sequence. *Developmental Science, 16*(6), 841-849.

Felisberti, F., & Terry, P. (2015). The effects of alcohol on the recognition of facial expressions and microexpressions of emotion: enhanced recognition of disgust and contempt. *Human Psychopharmacology: Clinical and Experimental, 30*(5), 384-392.

Field, M., Wiers, R. W., Christiansen, P., Fillmore, M. T., & Verster, J. C. (2010). Acute Alcohol Effects on Inhibitory Control and Implicit Cognition: Implications for Loss of Control Over Drinking. *Alcoholism-Clinical and Experimental Research, 34*(8), 1346-1352.

Finkel, E. J., DeWall, C. N., Slotter, E. B., McNulty, J. K., Pond, R. S., & Jr, A., D.C. (2012). Using I^3 theory to clarify when dispositional aggressiveness predicts intimate partner violence perpetration. *Journal of Personality and Social Psychology, 102*(2), 533–549.

Fiske, A. P., & Haslam, N. (1996). Social cognition is thinking about relationships. *Current Directions in Psychological Science, 5*, 143–148.

Floyd, K., & Erbert, L. A. (2003). Relational message interpretations of nonverbal matching behavior: an application of the social meaning model. *The Journal of Social Psychology, 143*(5), 581-597.

Foisy, M. L., Kornreich, C., Fobe, A., D'Hondt, L., Pelc, I., Hanak, C., Verbanck, P., & Philippot, P. (2007). Impaired emotional facial expression recognition in alcohol dependence: Do these deficits persist with midterm abstinence? *Alcoholism-Clinical and Experimental Research, 31*(3), 404-410.

Foran, H. M., & O'Leary, K. D. (2008). Alcohol and intimate partner violence: a meta-analytic review. *Clinical Psychology Review, 28*(7), 1222-1234.

Forgays, D. G., Forgays, D. K., & Spielberger, C. D. (1997). Factor structure of the State-Trait Anger Expression Inventory. *Journal of Personality Assessment, 69*(3), 497-507.

Frigerio, E., Burt, D. M., Montagne, B., Murray, L. K., & Perrett, D. I. (2002). Facial affect perception in alcoholics. *Psychiatry Research, 113*(1-2), 161-171.

Gallagher, K. E., Hudepohl, A. D., & Parrott, D. J. (2010). Power of being present: the role of mindfulness on the relation between men's alcohol use and sexual aggression toward intimate partners. *Aggressive Behavior, 36*(6), 405-413.

Gallagher, K. E., & Parrott, D. J. (2011). Does distraction reduce the alcohol-aggression relation? A cognitive and behavioral test of the attention-allocation model. *Journal of Consulting and Clinical Psychology, 79*(3), 319-329.

Gallagher, K. E., & Parrott, D. J. (2016). A self-awareness intervention manipulation for heavy-drinking men's alcohol-related aggression toward women. *Journal of Consulting and Clinical Psychology, 84*(9), 813-823.

Ganguli, M., Vander Bilt, J., Saxton, J. A., Shen, C., & Dodge, H. H. (2005). Alcohol consumption and cognitive function in late life: a longitudinal community study. *Neurology, 65*(8), 1210-1217.

Geen, R. G. (2001). *Human aggression* (2nd ed. ed.). Open University.

Geen, R. G., & Donnerstein, E. I. (1983). *Aggression : theoretical and empirical reviews*. Academic.

Gery, I., Miljkovitch, R., Berthoz, S., & Soussignan, R. (2009). Empathy and recognition of facial expressions of emotion in sex offenders, non-sex offenders and normal controls. *Psychiatry Research, 165*(3), 252-262.

Giancola, P. R. (2000). Executive functioning: a conceptual framework for alcohol-related aggression. *Experimental and Clinical Psychopharmacology, 8*(4), 576-597.

Giancola, P. R. (2002a). Alcohol-related aggression in men and women: The influence of dispositional aggressivity. *Journal of Studies on Alcohol, 63*(6), 696-708.

Giancola, P. R. (2002b). The influence of trait anger on the alcohol-aggression relation in men and women. *Alcoholism: Clinical and Experimental Research, 26*(9), 1350-1358.

Giancola, P. R. (2002c). Irritability, acute alcohol consumption and aggressive behavior in men and women. *Drug and Alcohol Dependence, 68*(3), 263-274.

Giancola, P. R. (2003). The moderating effects of dispositional empathy on alcohol-related aggression in men and women. *Journal of Abnormal Psychology, 112*(2), 275-281.

Giancola, P. R., & Corman, M. D. (2007). Alcohol and aggression: a test of the attention-allocation model. *Psychological Science, 18*(7), 649-655.

Giancola, P. R., Duke, A. A., & Ritz, K. Z. (2011). Alcohol, violence, and the Alcohol Myopia Model: preliminary findings and implications for prevention. *Addictive Behaviors, 36*(10), 1019-1022.

Giancola, P. R., Godlaski, A. J., & Parrott, D. J. (2005). "So I can't blame the booze?": Dispositional aggressivity negates the moderating effects of expectancies on alcohol-related aggression. *Journal of Studies on Alcohol, 66*(6), 815-824.

Giancola, P. R., Helton, E. L., Osborne, A. B., Terry, M. K., Fuss, A. M., & Westerfield, J. A. (2002). The effects of alcohol and provocation on aggressive behavior in men and women. *Journal of Studies on Alcohol and Drugs, 63*(1), 64-73.

Giancola, P. R., Josephs, R. A., DeWall, C. N., & Gunn, R. L. (2009). Applying the attention-allocation model to the explanation of alcohol-related aggression: implications for prevention. *Substance Use & Misuse, 44*(9-10), 1263-1279.

Giancola, P. R., Josephs, R. A., Parrott, D. J., & Duke, A. A. (2010). Alcohol Myopia Revisited: Clarifying Aggression and Other Acts of Disinhibition Through a Distorted Lens. *Perspectives on Psychological Science, 5*(3), 265-278.

Giancola, P. R., Levinson, C. A., Corman, M. D., Godlaski, A. J., Morris, D. H., Phillips, J. P., & Holt, J. C. (2009). Men and women, alcohol and aggression. *Experimental and Clinical Psychopharmacology, 17*(3), 154-164.

Giancola, P. R., & Zeichner, A. (1995). Alcohol-Related Aggression in Males and Females - Effects of Blood-Alcohol Concentration, Subjective Intoxication, Personality, and Provocation. *Alcoholism-Clinical and Experimental Research, 19*(1), 130-134.

Gillespie, S. M., Rotshtein, P., Satherley, R. M., Beech, A. R., & Mitchell, I. J. (2015). Emotional expression recognition and attribution bias among sexual and violent offenders: a signal detection analysis. *Frontiers in Psychology, 6*, 595.

Gilligan, C., Anderson, K. G., Ladd, B. O., Yong, Y. M., & David, M. (2019). Inaccuracies in survey reporting of alcohol consumption. *BMC Public Health, 19*(1), 1639.

Gilman, J. M., Ramchandani, V. A., Davis, M. B., Bjork, J. M., & Hommer, D. W. (2008). Why we like to drink: a functional magnetic resonance imaging study of the rewarding and anxiolytic effects of alcohol. *Journal of Neuroscience Research, 28*(18), 4583-4591.

Goldman, M. S., Brown, S. A., & Christiansen, B. A. (1987). Expectancy theory: thinking about drinking. In H. T. Blane & K. E. Leonard (Eds.), *Psychological Theories of Drinking and Alcoholism* (pp. 181–226). Guilford.

Gosselin, P. (1995). The development of the recognition of facial expressions of emotion in children. *Canadian Journal of Behavioural Science, 27*, 107–119.

Gray, K., Schein, C., & Ward, A. F. (2014). The myth of harmless wrongs in moral cognition: Automatic dyadic completion from sin to suffering. *Journal of Experimental Psychology: General, 143*(4), 1600-1615.

Grier, J. B. (1971). Nonparametric indexes for sensitivity and bias: computing formulas. *Psychological Bulletin, 75*(6), 424-429.

Griffiths, S., Jarrold, C., Penton-Voak, I. S., & Munafo, M. R. (2015). Feedback training induces a bias for detecting happiness or fear in facial expressions that generalises to a novel task. *Psychiatry Research, 230*(3), 951-957.

Gual, A., Angel Arbesu, J., Zarco, J., Balcells-Olivero, M. L. M., Lopez-Pelayo, H., Miquel, L., & Bobes, J. (2017). Risky Drinkers Underestimate their Own Alcohol Consumption. *Alcohol Alcohol, 52*(4), 516-517.

Gustafson, R. (1985). Frustration as an important determinant of alcohol-related aggression. *Psychological Reports, 57*(1), 3-14.

Haggard-Grann, U., Hallqvist, J., Langstrom, N., & Moller, J. (2006). The role of alcohol and drugs in triggering criminal violence: a case-crossover study*. *Addiction, 101*(1), 100-108.

Hall, C. W. (2006). Self-reported aggression and the perception of anger in facial expression photos (vol 140, pg 261, 2006). *Journal of Psychology, 140*(4), 395-396.

Hamlin, J. K. (2013). Failed attempts to help and harm: intention versus outcome in preverbal infants' social evaluations. *Cognition, 128*(3), 451-474.

Harmer, C. J., Thilo, K. V., Rothwell, J. C., & Goodwin, G. M. (2001). Transcranial magnetic stimulation of medial-frontal cortex impairs the processing of angry facial expressions. *Nature Neuroscience, 4*(1), 17-18.

Harris, G. J., Jaffin, S. K., Hodge, S. M., Kennedy, D., Caviness, V. S., Marinkovic, K., Papadimitriou, G. M., Makris, N., & Oscar-Berman, M. (2008). Frontal white matter and cingulum diffusion tensor imaging deficits in alcoholism. *Alcoholism: Clinical and Experimental Research, 32*(6), 1001-1013.

Hart, S. (2011). *The Impact of Attachment*. W. W. Norton & Co.

Heale, R., & Forbes, D. (2013). Understanding triangulation in research. *Evidence-Based Nursing, 16*(4), 98.

Henderson, S. E., Vallejo, A. I., Ely, B. A., Kang, G., Krain Roy, A., Pine, D. S., Stern, E. R., & Gabbay, V. (2014). The neural correlates of emotional face-processing in adolescent depression: a dimensional approach focusing on anhedonia and illness severity. *Psychiatry Research, 224*(3), 234-241.

Herba, C., & Phillips, M. (2004). Annotation: Development of facial expression recognition from childhood to adolescence: behavioural and neurological perspectives. *Journal of Child Psychology and Psychiatry, 45*(7), 1185-1198.

Hertenstein, M. J., & Campos, J. J. (2004). The retention effects of an adult's emotional displays on infant behavior. *Child development, 75*(2), 595-613.

Heuer, K., Rinck, M., & Becker, E. S. (2007). Avoidance of emotional facial expressions in social anxiety: The Approach-Avoidance Task. *Behaviour Research and Therapy, 45*(12), 2990-3001.

Hoaken, P. N. S., Allaby, D. B., & Earle, J. (2007). Executive cognitive functioning and the recognition of facial expressions of emotion in incarcerated violent offenders, non-violent offenders, and controls. *Aggressive Behavior, 33*(5), 412-421.

Hoaken, P. N. S., Shaughnessy, V. K., & Pihl, R. O. (2003). Executive Cognitive Functioning and Aggression: Is It a Issue of Impulsivity? *Aggressive Behavior, 29*, 15-30.

Hoaken, P. N. S., & Stewart, S. H. (2003). Drugs of abuse and the elicitation of human aggressive behavior. *Addictive Behaviors, 28*(9), 1533-1554.

Hornak, J., Rolls, E. T., & Wade, D. (1996). Face and voice expression identification in patients with emotional and behavioural changes following ventral frontal lobe damage. *Neuropsychologia, 34*(4), 247-261.

Intille, S., Haynes, C., Maniar, D., Ponnada, A., & Manjourides, J. (2016). muEMA: Microinteraction-based Ecological Momentary Assessment (EMA) Using a Smartwatch. *Proc ACM Int Conf Ubiquitous Comput, 2016*, 1124-1128.

Ito, T. A., Miller, N., & Pollock, V. E. (1996). Alcohol and aggression: a meta-analysis on the moderating effects of inhibitory cues, triggering events, and self-focused attention. *Psychological Bulletin, 120*(1), 60-82.

Izard, C., Fine, S., Schultz, D., Mostow, A., Ackerman, B., & Youngstrom, E. (2001). Emotion knowledge as a predictor of social behavior and academic competence in children at risk. *Psychological Science, 12*(1), 18-23.

Joormann, J., & Gotlib, I. H. (2006). Is this happiness I see? Biases in the identification of emotional facial expressions in depression and social phobia. *Journal of Abnormal Psychology, 115*(4), 705-714.

Kano, M., Gyoba, J., Kamachi, M., Mochizuki, H., Hongo, M., & Yanai, K. (2003). Low doses of alcohol have a selective effect on the recognition of happy facial expressions. *Human Psychopharmacology-Clinical and Experimental, 18*(2), 131-139.

Khouja, J. N., Attwood, A. S., Penton-Voak, I. S., & Munafo, M. R. (2019). Effects of acute alcohol consumption on emotion recognition in social alcohol drinkers. *Journal of Psychopharmacology, 33*(3), 326-334.

Kimonis, E. R., Frick, P. J., Fazekas, H., & Loney, B. R. (2006). Psychopathy, aggression, and the processing of emotional stimuli in non-referred girls and boys. *Behavioral Sciences & the Law, 24*(1), 21-37.

Klinnert, M. D. (1983). Emotional Expressions as Behavior Regulators. In R. Plutchik & H. Kellerman (Eds.), *Emotion: Theory, Research, and Experience* (Vol. 2). Academic Press.

Ko, B. C. (2018). A Brief Review of Facial Emotion Recognition Based on Visual Information. *Sensors, 18*(2).

Kornreich, C., Blairy, S., Philippot, P., Dan, B., Foisy, M., Hess, U., Le Bon, O., Pelc, I., & Verbanck, P. (2001). Impaired emotional facial expression recognition in alcoholism compared with obsessive-compulsive disorder and normal controls. *Psychiatry Research, 102*(3), 235-248.

Kornreich, C., Foisy, M. L., Philippot, P., Dan, B., Tecco, J., Noel, X., Hess, U., Pelc, I., & Verbanck, P. (2003). Impaired emotional facial expression recognition in alcoholics, opiate dependence subjects, methadone maintained subjects and mixed alcohol-opiate antecedents subjects compared with normal controls. *Psychiatry Research, 119*(3), 251-260.

Kornreich, C., Philippot, P., Foisy, M. L., Blairy, S., Raynaud, E., Dan, B., Hess, U., Noel, X., Pelc, I., & Verbanck, P. (2002). Impaired emotional facial expression recognition is associated with interpersonal problems in alcoholism. *Alcohol Alcohol, 37*(4), 394-400.

Kosson, D. S., Suchy, Y., Mayer, A. R., & Libby, J. (2002). Facial affect recognition in criminal psychopaths. *Emotion, 2*(4), 398-411.

Krieglmeyer, R., & Deutsch, R. (2010). Comparing measures of approach-avoidance behaviour: the manikin task vs. two versions of the joystick task. *Cognition & Emotion, 24*, 810–828.

Latif, N., Barbosa, A. V., Vatikiotis-Bateson, E., Castelhano, M. S., & Munhall, K. G. (2014). Movement coordination during conversation. *PLoS One, 9*(8), e105036.

Lawlor, D. A., Tilling, K., & Davey Smith, G. (2016). Triangulation in aetiological epidemiology. *International Journal of Epidemiology, 45*(6), 1866-1886.

Leonard, K. E., & Blane, H. T. (1992). Alcohol and marital aggression in a national sample of young men. *Journal of Interpersonal Violence, 7*, 19-30.

Leppanen, J. M., Milders, M., Bell, J. S., Terriere, E., & Hietanen, J. K. (2004). Depression biases the recognition of emotionally neutral faces. *Psychiatry Research, 128*(2), 123-133.

Lipsey, M. W., Wilson, D. B., Cohen, M. A., & Derzon, J. H. (1997). Is there a causal relationship between alcohol use and violence? A synthesis of evidence. *Recent Developments in Alcoholism, 13*, 245-282.

Luke, S. G. (2017). Evaluating significance in linear mixed-effects models in R. *Behavior Research Methods, 49*(4), 1494-1502.

Lundholm, L., Haggard, U., Moller, J., Hallqvist, J., & Thiblin, I. (2013). The triggering effect of alcohol and illicit drugs on violent crime in a remand prison population: a case crossover study. *Drug and Alcohol Dependence, 129*(1-2), 110-115.

MacDonald, T. K., Fong, G. T., Zanna, M. P., & Martineau, A. M. (2000). Alcohol myopia and condom use: can alcohol intoxication be associated with more prudent behavior? *Journal of Personality and Social Psychology, 78*(4), 605-619.

Macmillan, N. A., & Creelman, C. D. (2005). *Detection theory : a user's guide* (2nd ed.). Lawrence Erlbaum Associates. Publisher description http://www.loc.gov/catdir/enhancements/fy0644/2004043261-d.html
Table of contents only http://www.loc.gov/catdir/enhancements/fy0644/2004043261-t.html

Macrae, C. N., & Quadflieg, S. (2010). Perceiving people. In S. Fiske, D. T. Gilbert, & G. Lindzey (Eds.), *The Handbook of Social Psychology* (5th ed., pp. 428–463). McGraw-Hill.

Mahedy, L., Suddell, S., Skirrow, C., Fernandes, G. S., Field, M., Heron, J., Hickman, M., Wootton, R., & Munafo, M. R. (2020). Alcohol use and cognitive functioning in young adults: improving causal inference. *Addiction*.

Makris, N., Oscar-Berman, M., Jaffin, S. K., Hodge, S. M., Kennedy, D. N., Caviness, V. S., Marinkovic, K., Breiter, H. C., Gasic, G. P., & Harris, G. J. (2008). Decreased volume of the brain reward system in alcoholism. *Biological Psychiatry, 64*(3), 192-202.

Mancini, G., Biolcati, R., Agnoli, S., Andrei, F., & Trombini, E. (2018). Recognition of Facial Emotional Expressions Among Italian Pre-adolescents, and Their Affective Reactions. *Frontiers in Psychology, 9*, 1303.

Marczinski, C. A., Abroms, B. D., Van Selst, M., & Fillmore, M. T. (2005). Alcohol-induced impairment of behavioral control: differential effects on engaging vs. disengaging responses. *Psychopharmacology, 182*(3), 452-459.

Marczinski, C. A., & Fillmore, M. T. (2005). Alcohol increases reliance on cues that signal acts of control. *Experimental and Clinical Psychopharmacology, 13*(1), 15-24.

Marinkovic, K., Oscar-Berman, M., Urban, T., O'Reilly, C. E., Howard, J. A., Sawyer, K., & Harris, G. J. (2009). Alcoholism and dampened temporal limbic activation to emotional faces. *Alcoholism: Clinical and Experimental Research, 33*(11), 1880-1892.

Marsh, A. A., Ambady, N., & Kleck, R. E. (2005). The effects of fear and anger facial expressions on approach- and avoidance-related behaviors. *Emotion, 5*(1), 119-124.

Marsh, A. A., Kozak, M. N., & Ambady, N. (2007). Accurate identification of fear facial expressions predicts prosocial behavior. *Emotion, 7*(2), 239-251.

Martin, C. S., Earleywine, M., Musty, R. E., Perrine, M. W., & Swift, R. M. (1993). Development and Validation of the Biphasic Alcohol Effects Scale. *Alcoholism-Clinical and Experimental Research, 17*(1), 140-146.

Marwick, K., & Hall, J. (2008). Social cognition in schizophrenia: a review of face processing. *British Medical Bulletin, 88*(1), 43-58.

Mast, S. M., & Hall, J. A. (2004). Who is the boss and who is not? Accuracy of judging status. *Journal of Nonverbal Behavior, 28*, 145–165.

Maurage, P., Campanella, S., Philippot, P., de Timary, P., Constant, E., Gauthier, S., Micciche, M. L., Kornreich, C., Hanak, C., Noel, X., & Verbanck, P. (2008). Alcoholism leads to early perceptive alterations, independently of comorbid depressed state: an ERP study. *Clinical Neurophysiology, 38*(2), 83-97.

Maurage, P., Campanella, S., Philippot, P., Martin, S., & de Timary, P. (2008). Face processing in chronic alcoholism: a specific deficit for emotional features. *Alcoholism: Clinical and Experimental Research, 32*(4), 600-606.

Maurage, P., Campanella, S., Philippot, P., Pham, T. H., & Joassin, F. (2007). The crossmodal facilitation effect is disrupted in alcoholism: a study with emotional stimuli. *Alcohol Alcohol, 42*(6), 552-559.

Maurage, P., Campanella, S., Philippot, P., Vermeulen, N., Constant, E., Luminet, O., & de Timary, P. (2008). Electrophysiological correlates of the disrupted processing of anger in alcoholism. *International Journal of Psychophysiology, 70*(1), 50-62.

Maurage, P., Philippot, P., Joassin, F., Pauwels, L., Pham, T., Prieto, E. A., Palmero-Soler, E., Zanow, F., & Campanella, S. (2008). The auditory-visual integration of anger is impaired in alcoholism: an event-related potentials study. *Journal of Psychiatry & Neuroscience, 33*(2), 111-122.

Maurage, P., Philippot, P., Verbanck, P., Noel, X., Kornreich, C., Hanak, C., & Campanella, S. (2007). Is the P300 deficit in alcoholism associated with early visual impairments (P100, N170)? An oddball paradigm. *Clinical Neurophysiology, 118*(3), 633-644.

McClelland, G. M., & Teplin, L. A. (2001). Alcohol intoxication and violent crime: implications for public health policy. *The American Journal on Addictions, 10*(s1), s70-s85.

Michael, J., Sebanz, N., & Knoblich, G. (2016). Observing joint action: Coordination creates commitment. *Cognition, 157*, 106-113.

Milich, R., & Dodge, K. A. (1984). Social information processing in child psychiatric populations. *Journal of Abnormal Child Psychology, 12*(3), 471-489.

Milinski, M. (2016). Reputation, a universal currency for human social interactions. *Philosophical Transactions of the Royal Society B: Biological Sciences, 371*(1687), 20150100.

Miller, C. A., Parrott, D. J., & Giancola, P. R. (2009). Agreeableness and Alcohol-Related Aggression: The Mediating Effect of Trait Aggressivity. *Experimental and Clinical Psychopharmacology, 17*(6), 445-455.

Miller, M. A., Bershad, A. K., & de Wit, H. (2015). Drug effects on responses to emotional facial expressions: recent findings. *Behavioural Pharmacology, 26*(6), 571-579.

Moeller, F. G., Dougherty, D. M., Lane, S. D., Steinberg, J. L., & Cherek, D. R. (1998). Antisocial personality disorder and alcohol-induced aggression. *Alcoholism-Clinical and Experimental Research, 22*(9), 1898-1902.

Mohlman, J., Carmin, C. N., & Price, R. B. (2007). Jumping to interpretations: social anxiety disorder and the identification of emotional facial expressions. *Behaviour Research and Therapy, 45*(3), 591-599.

Monahan, J. L., & Lannutti, P. J. (2000). Alcohol as social lubricant: Alcohol myopia theory, social self-esteem, and social interaction. *Human Communication Research, 26*(2), 175–202.

Moriya, J., Tanno, Y., & Sugiura, Y. (2013). Repeated short presentations of morphed facial expressions change recognition and evaluation of facial expressions. *Psychological Research-Psychologische Forschung, 77*(6), 698-707.

Morris, J. S., Frith, C. D., Perrett, D. I., Rowland, D., Young, A. W., Calder, A. J., & Dolan, R. J. (1996). A differential neural response in the human amygdala to fearful and happy facial expressions. *Nature, 383*(6603), 812-815.

Murdoch, D., Pihl, R. O., & Ross, D. (1990). Alcohol and crimes of violence: present issues. *International Journal of Mental Health and Addiction, 25*(9), 1065-1081.

Nasby, W., Hayden, B., & DePaulo, B. M. (1980). Attributional bias among aggressive boys to interpret unambiguous social stimuli as displays of hostility. *Journal of Abnormal Psychology, 89*(3), 459-468.

Neafsey, E. J., & Collins, M. A. (2011). Moderate alcohol consumption and cognitive risk. *Neuropsychiatric Disease and Treatment, 7*, 465-484.

Newberry, M., Williams, N., & Caulfield, L. (2013). Female alcohol consumption, motivations for aggression and aggressive incidents in licensed premises. *Addictive Behaviors, 38*(3), 1844-1851.

NHS. (2018, 13 April 2018). *Alcohol units, NHS UK*. NHS UK. https://www.nhs.uk/live-well/alcohol-support/calculating-alcohol-units/

O'Daly, O. G., Trick, L., Scaife, J., Marshall, J., Ball, D., Phillips, M. L., Williams, S. S., Stephens, D. N., & Duka, T. (2012). Withdrawal-associated increases and decreases in functional neural connectivity associated with altered emotional regulation in alcoholism. *Neuropsychopharmacology, 37*(10), 2267-2276.

Office for National Statistics. (2018). *Data on alcohol related incidents, years ending March 2011 to March 2017, Crime Survey for England and Wales*. Retrieved 01/07/2020 from https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/adhocs/009372dataonalcoholrelatedincidentsyearsendingmarch2011tomarch2017crimesurveyforenglandandwales

Office for National Statistics. (2019). *Nature of crime tables, violence; Year ending March 2018*. Retrieved 01/07/2020 from https://www.ons.gov.uk/peoplepopulationandcommunity/crimeandjustice/datasets/natureofcrimetablesviolence

Ohman, A. (2005). The role of the amygdala in human fear: automatic detection of threat. *Psychoneuroendocrinology, 30*(10), 953-958.

Ohman, A., & Mineka, S. (2001). Fears, phobias, and preparedness: toward an evolved module of fear and fear learning. *Psychological Review, 108*(3), 483-522.

Oscar-Berman, M., & Marinkovic, K. (2007). Alcohol: effects on neurobehavioral functions and the brain. *Neuropsychology Review, 17*(3), 239-257.

Oyegbile, T. O., & Marler, C. A. (2006). Weak winner effect in a less aggressive mammal: correlations with corticosterone but not testosterone. *Physiology & Behavior, 89*(2), 171-179.

Paglia, A., & Room, R. (2006). Expectancies About the Effects of Alcohol on the Self and on Others as Determinants of Alcohol Policy Attitudes. *Journal of Applied Social Psychology, 29*, 2632–2651.

Parrott, D. J., & Eckhardt, C. I. (2018). Effects of Alcohol on Human Aggression. *Current Opinion in Psychology, 19*, 1-5.

Parrott, D. J., Gallagher, K. E., & Zeichner, A. (2012). Liquid courage or liquid fear: alcohol intoxication and anxiety facilitate physical aggression. *Substance Use & Misuse, 47*(7), 774-786.

Parrott, D. J., & Giancola, P. R. (2004). A further examination of the relation between trait anger and alcohol-related aggression: the role of anger control. *Alcoholism: Clinical and Experimental Research, 28*(6), 855-864.

Parrott, D. J., & Zeichner, A. (2002). Effects of alcohol and trait anger on physical aggression in men. *Journal of Studies on Alcohol and Drugs, 63*(2), 196-204.

Pawliczek, C. M., Derntl, B., Kellermann, T., Kohn, N., Gur, R. C., & Habel, U. (2013). Inhibitory control and trait aggression: neural and behavioral insights using the emotional stop signal task. *Neuroimage, 79*, 264-274.

Penton-Voak, I. S., Thomas, J., Gage, S. H., McMurran, M., McDonald, S., & Munafo, M. R. (2013). Increasing recognition of happiness in ambiguous facial expressions reduces anger and aggressive behavior. *Psychological Science, 24*(5), 688-697.

Pernanen, K. (1991). *Alcohol in human violence*. Guilford Press.

Perugini, M., Gallucci, M., & Costantini, G. (2018). A Practical Primer To Power Analysis for Simple Experimental Designs. *International Review of Social Psychology, 31*(1), 20.

Phaf, R. H., Mohr, S. E., Rotteveel, M., & Wicherts, J. M. (2014). Approach, avoidance, and affect: a meta-analysis of approach-avoidance tendencies in manual reaction time tasks. *Frontiers in Psychology, 5*, 378.

Philippot, P., Kornreich, C., Blairy, S., Baert, I., Den Dulk, A., Le Bon, O., Streel, E., Hess, U., Pelc, I., & Verbanck, P. (1999). Alcoholics' deficits in the decoding of emotional facial expression. *Alcoholism-Clinical and Experimental Research, 23*(6), 1031-1038.

Pihl, R. O., & Sutton, R. (2009). Drugs and aggression readily mix; so what now? *Substance Use & Misuse, 44*(9-10), 1188-1203.

Place, S. S., Todd, P. M., Penke, L., & Asendorpf, J. B. (2009). The ability to judge the romantic interest of others. *Psychological Science, 20*(1), 22-26.

Pollack, I., & Norman, D. A. (1964). A Non-Parametric Analysis of Recognition Experiments. *Psychonomic Science, 1*(5), 125-126.

Puiu, A. A., Wudarczyk, O., Goerlich, K. S., Votinov, M., Herpertz-Dahlmann, B., Turetsky, B., & Konrad, K. (2018). Impulsive aggression and response inhibition in attention-deficit/hyperactivity disorder and disruptive behavioral disorders: Findings from a systematic review. *Neuroscience & Biobehavioral Reviews, 90*, 231-246.

Purvis, D. M., Gallagher, K. E., & Parrott, D. J. (2016). Reducing alcohol-related aggression: Effects of a self-awareness manipulation and locus of control in heavy drinking males. *Addictive Behaviors, 58*, 31-34.

Quadflieg, S., & Penton-Voak, I. S. (2017). The Emerging Science of People-Watching: Forming Impressions From Third-Party Encounters. *Current Directions in Psychological Science, 26*(4), 383-389.

Quadflieg, S., & Westmoreland, K. (2019). Making Sense of Other People's Encounters: Towards an Integrative Model of Relational Impression Formation. *Journal of Nonverbal Behavior 43*, 233–256.

Quinn, P. D., Stappenbeck, C. A., & Fromme, K. (2013). An event-level examination of sex differences and subjective intoxication in alcohol-related aggression. *Experimental and Clinical Psychopharmacology, 21*(2), 93-102.

R Core Team. (2020). *R: A Language and Environment for Statistical Computing.* In (Version 3.6.3) R Foundation for Statistical Computing. https://www.R-project.org/

R Studio. (2019). *RStudio: Integrated Development for R.* In RStudio, Inc. http://www.rstudio.com/.

Raaijmakers, M. A., Smidts, D. P., Sergeant, J. A., Maassen, G. H., Posthumus, J. A., van Engeland, H., & Matthys, W. (2008). Executive functions in preschool children with aggressive behavior: impairments in inhibitory control. *Journal of Abnormal Child Psychology, 36*(7), 1097-1107.

Ramírez, J. M., & Andreu, J. M. (2006). Aggression, and some related psychological constructs (anger, hostility, and impulsivity); some comments from a research project. *Neuroscience & Biobehavioral Reviews, 30*(3), 276–291.

Robertson, K., Forbes, S., & Thyne, M. (2020). Perpetration of Alcohol-Related Aggression by Male and Female College Students: An Examination of Overt and Relational Aggression. *Journal of Interpersonal Violence, 35*(5-6), 1454-1475.

Robinson, L., Spencer, M. D., Thomson, L. D., Sprengelmeyer, R., Owens, D. G., Stanfield, A. C., Hall, J., Baig, B. J., MacIntyre, D. J., McKechanie, A., & Johnstone, E. C. (2012). Facial emotion recognition in Scottish prisoners. *International Journal of Law and Psychiatry, 35*(1), 57-61.

Romanski, L. M., & LeDoux, J. E. (1992). Equipotentiality of thalamo-amygdala and thalamo-cortico-amygdala circuits in auditory fear conditioning. *Journal of Neuroscience Research, 12*(11), 4501-4509.

Rule, R. R., Shimamura, A. P., & Knight, R. T. (2002). Orbitofrontal cortex and dynamic filtering of emotional stimuli. *Cognitive, Affective, & Behavioral Neuroscience, 2*(3), 264-270.

Salloum, J. B., Ramchandani, V. A., Bodurka, J., Rawlings, R., Momenan, R., George, D., & Hommer, D. W. (2007). Blunted rostral anterior cingulate response during a simplified decoding task of negative emotional facial expressions in alcoholic patients. *Alcoholism: Clinical and Experimental Research, 31*(9), 1490-1504.

Sarkola, T., & Eriksson, C. J. (2003). Testosterone increases in men after a low dose of alcohol. *Alcoholism: Clinical and Experimental Research, 27*(4), 682-685.

Sarkola, T., Fukunaga, T., Makisalo, H., & Peter Eriksson, C. J. (2000). Acute effect of alcohol on androgens in premenopausal women. *Alcohol Alcohol, 35*(1), 84-90.

Saunders, J. B., Aasland, O. G., Babor, T. F., Delafuente, J. R., & Grant, M. (1993). Development of the Alcohol-Use Disorders Identification Test (Audit) - Who Collaborative Project on Early Detection of Persons with Harmful Alcohol-Consumption .2. *Addiction, 88*(6), 791-804.

Sayette, M. A. (1993). An appraisal-disruption model of alcohol's effects on stress responses in social drinkers. *Psychological Bulletin, 114*(3), 459-476.

Sayette, M. A., Breslin, F. C., Wilson, G. T., & Rosenblum, G. D. (1994). An Evaluation of the Balanced Placebo Design in Alcohol Administration Research. *Addictive Behaviors, 19*(3), 333-342.

Sayette, M. A., Martin, C. S., Perrott, M. A., Wertz, J. M., & Hufford, M. R. (2001). A test of the appraisal-disruption model of alcohol and stress. *Journal of Studies on Alcohol and Drugs, 62*(2), 247-256.

Schonenberg, M., Christian, S., Gausser, A. K., Mayer, S. V., Hautzinger, M., & Jusyte, A. (2014). Addressing perceptual insensitivity to facial affect in violent offenders: first evidence for the efficacy of a novel implicit training approach. *Psychological Medicine, 44*(5), 1043-1052.

Schonenberg, M., & Jusyte, A. (2014). Investigation of the hostile attribution bias toward ambiguous facial cues in antisocial violent offenders. *European Archives of Psychiatry and Clinical Neuroscience, 264*(1), 61-69.

Schonenberg, M., Louis, K., Mayer, S., & Jusyte, A. (2013). Impaired identification of threat-related social information in male delinquents with antisocial personality disorder. *Journal of Personality Disorders, 27*(4), 496-505.

Scott, K. D., Schafer, J., & Greenfield, T. K. (1999). The role of alcohol in physical assault perpetration and victimization. *Journal of Studies on Alcohol and Drugs, 60*(4), 528-536.

Seidel, E. M., Habel, U., Kirschner, M., Gur, R. C., & Derntl, B. (2010). The impact of facial emotional expressions on behavioral tendencies in women and men. *Journal of Experimental Psychology: Human Perception and Performance, 36*(2), 500-507.

Seidel, E. M., Pfabigan, D. M., Keckeis, K., Wucherer, A. M., Jahn, T., Lamm, C., & Derntl, B. (2013). Empathic competencies in violent offenders. *Psychiatry Research, 210*(3), 1168-1175.

Setia, M. S. (2016). Methodology Series Module 3: Cross-sectional Studies. *Indian Journal of Dermatology, 61*(3), 261-264.

Sinzig, J., Walter, D., & Doepfner, M. (2009). Attention deficit/hyperactivity disorder in children and adolescents with autism spectrum disorder: symptom or syndrome? *Journal of Attention Disorders, 13*(2), 117-126.

Skinner, A. L., & Benton, C. P. (2010). Anti-expression aftereffects reveal prototype-referenced coding of facial expressions. *Psychological Science, 21*(9), 1248-1253.

Slotter, E. B., & Finkel, E. J. (2011). I³ theory: Instigating, impelling, and inhibiting factors in aggression. In P. R. Shaver & M. Mikulincer (Eds.), *Herzilya series on personality and social psychology. Human aggression and violence: Causes, manifestations, and consequences* American Psychological Association. https://doi.org/https://doi.org/10.1037/12346-002

Smeijers, D., Rinck, M., Bulten, E., van den Heuvel, T., & Verkes, R. J. (2017). Generalized hostile interpretation bias regarding facial expressions: Characteristic of pathological aggressive behavior. *Aggressive Behavior, 43*(4), 386-397.

Smith, P., & Waterman, M. (2004). Role of experience in processing bias for aggressive words in forensic and non-forensic populations. *Aggressive Behavior, 30*, 105-122.

Soma, K. K. (2006). Testosterone and aggression: Berthold, birds and beyond. *Journal of Neuroendocrinology, 18*(7), 543-551.

Spielberger, C. D. (1999). *State-Trait Anger Expression Inventory-2 (STAXI-2). Professional Manual*. Psychological Assessment Resources https://doi.org/10.1002/9780470479216.corpsy0942

Sripada, C. S., Angstadt, M., McNamara, P., King, A. C., & Phan, K. L. (2011). Effects of alcohol on brain responses to social signals of threat in humans. *Neuroimage, 55*(1), 371-380.

Stampfer, M. J., Kang, J. H., Chen, J., Cherry, R., & Grodstein, F. (2005). Effects of moderate alcohol consumption on cognitive function in women. *The New England Journal of Medicine, 352*(3), 245-253.

Stanislaw, H., & Todorov, N. (1999). Calculation of signal detection theory measures. *Behavior Research Methods, 31*(1), 137-149.

Stappenbeck, C. A., & Fromme, K. (2010). Alcohol use and perceived social and emotional consequences among perpetrators of general and sexual aggression. *Journal of Interpersonal Violence, 25*(4), 699-715.

Steele, C. M., & Josephs, R. A. (1990). Alcohol myopia. Its prized and dangerous effects. *American Psychologist, 45*(8), 921-933.

Steele, C. M., & Southwick, L. (1985). Alcohol and social behavior I: The psychology of drunken excess. *Journal of Personality and Social Psychology, 48*(1), 18-34.

Sterne, J. A., & Davey Smith, G. (2001). Sifting the evidence-what's wrong with significance tests? *BMJ, 322*(7280), 226-231.

Sterzer, P., Stadler, C., Krebs, A., Kleinschmidt, A., & Poustka, F. (2005). Abnormal neural responses to emotional visual stimuli in adolescents with conduct disorder. *Biological Psychiatry, 57*(1), 7-15.

Subra, B., Muller, D., Begue, L., Bushman, B. J., & Delmas, F. (2010). Automatic effects of alcohol and aggressive cues on aggressive thoughts and behaviors. *Personality and Social Psychology Bulletin, 36*(8), 1052-1057.

Suchy, Y. (2009). Executive functioning: overview, assessment, and research issues for non-neuropsychologists. *Annals of Behavioral Medicine, 37*(2), 106-116.

Swets, J. A. (1986). Form of Empirical Rocs in Discrimination and Diagnostic Tasks - Implications for Theory and Measurement of Performance. *Psychological Bulletin, 99*(2), 181-198.

Taylor, S. P. (1967). Aggressive Behavior and Physiological Arousal as a Function of Provocation and the Tendency to Inhibit Aggression. *Journal of Personality, 35*(2), 297-310.

Tedeschi, J. T., & Felson, R. B. (1994). *Violence, aggression & coercive actions*. American Psychological Association.

Testa, M. (2002). The impact of men's alcohol consumption on perpetration of sexual aggression. *Clinical Psychology Review, 22*(8), 1239-1263.

Testa, M., Fillmore, M. T., Norris, J., Abbey, A., Curtin, J. J., Leonard, K. E., Mariano, K. A., Thomas, M. C., Nomensen, K. J., George, W. H., VanZile-Tamsen, C., Livingston, J. A., Saenz, C., Buck, P. O., Zawacki, T., Parkhill, M. R., Jacques, A. J., & Hayman, L. W. (2006). Understanding alcohol expectancy effects: Revisiting the placebo condition. *Alcoholism-Clinical and Experimental Research, 30*(2), 339-348.

Theurel, A., Witt, A., Malsert, J., Lejeune, F., Fiorentini, C., Barisnikov, K., & Gentaz, E. (2016). The integration of visual context information in facial emotion recognition in 5- to 15-year-olds. *Journal of Experimental Child Psychology, 150*, 252-271.

Tiddeman, B., Burt, M., & Perrett, D. (2001). Prototyping and transforming facial textures for perception research. *IEEE Computer Graphics and Applications, 21*(5), 42-50.

Townshend, J. M., & Duka, T. (2003). Mixed emotions: alcoholics' impairments in the recognition of specific emotional facial expressions. *Neuropsychologia, 41*(7), 773-782.

Tremblay, P. F., Graham, K., & Wells, S. (2008). Severity of physical aggression reported by university students: A test of the interaction between trait aggression and alcohol consumption. *Personality and Individual Differences, 45*(1), 3-9.

Tsikandilakis, M., Bali, P., Derrfuss, J., & Chapman, P. (2020). Anger and hostility: are they different? An analytical exploration of facial-expressive differences, and physiological and facial-emotional responses. *Cognition & Emotion, 34*(3), 581-595.

Tucker, J. A., & Vuchinich, R. E. (1983). An Information-Processing Analysis of the Effects of Alcohol on Perceptions of Facial Emotions. *Psychopharmacology, 79*(2-3), 215-219.

Uekermann, J., & Daum, I. (2008). Social cognition in alcoholism: a link to prefrontal cortex dysfunction? *Addiction, 103*(5), 726-735.

Uleman, J. S., & Saribay, S. A. (2012). Initial impressions of others. In K. Deaux & M. Snyder (Eds.), *Oxford handbook of personality and social psychology* (pp. 337–366). Oxford University Press.

Van Bockstaele, B., van der Molen, M. J., van Nieuwenhuijzen, M., & Salemink, E. (2020). Modification of hostile attribution bias reduces self-reported reactive aggressive behavior in adolescents. *Journal of Experimental Child Psychology, 194*, 104811.

van Honk, J., Tuiten, A., Hermans, E., Putman, P., Koppeschaar, H., Thijssen, J., Verbaten, R., & van Doornen, L. (2001). A single administration of testosterone induces cardiac accelerative responses to angry faces in healthy young women. *Behavioral Neuroscience, 115*(1), 238-242.

Veenstra, L., Schneider, I. K., Bushman, B. J., & Koole, S. L. (2017). Drawn to danger: trait anger predicts automatic approach behaviour to angry faces. *Cognition & Emotion, 31*(4), 765-771.

Vogel-Sprott, M. (1967). Alcohol effects on human behaviour under reward and punishment. *Psychopharmacologia, 11*(4), 337-344.

Wang, L., McCarthy, G., Song, A. W., & Labar, K. S. (2005). Amygdala activation to sad pictures during high-field (4 tesla) functional magnetic resonance imaging. *Emotion, 5*(1), 12-22.

Watson, D., Clark, L. A., & Tellegen, A. (1988). Development and Validation of Brief Measures of Positive and Negative Affect - the Panas Scales. *Journal of Personality and Social Psychology, 54*(6), 1063-1070.

Wegrzyn, M., Westphal, S., & Kissler, J. (2017). In your face: the biased judgement of fear-anger expressions in violent offenders. *BMC Psychology, 5*(1), 16.

Wells, S., Graham, K., & West, P. (2000). Alcohol-related aggression in the general population. *Journal of Studies on Alcohol, 61*(4), 626-632.

Wiers, R. W., Eberl, C., Rinck, M., Becker, E. S., & Lindenmeyer, J. (2011). Retraining automatic action tendencies changes alcoholic patients' approach bias for alcohol and improves treatment outcome. *Psychological Science, 22*(4), 490-497.

Wieser, M. J., & Brosch, T. (2012). Faces in context: a review and systematization of contextual influences on affective face processing. *Frontiers in Psychology, 3*, 471.

Wilkowski, B. M., & Meier, B. P. (2010). Bring It On: Angry Facial Expressions Potentiate Approach-Motivated Motor Behavior. *Journal of Personality and Social Psychology, 98*(2), 496-505.

Wirth, M. M., & Schultheiss, O. C. (2007). Basal testosterone moderates responses to anger faces in humans. *Physiology & Behavior, 90*(2-3), 496-505.